A Dissertation on

"ASSESSMENT OF HEARING LOSS IN HIGH RISK INDIVIDUALS USING HIGH FREQUENCY PURE TONE AUDIOMETRY"

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI

With partial fulfillment of the regulations
For the award of the degree of

M.S. OTORHINOLARYNGOLOGY

BRANCH-IV

UPGRADED INSTITUTE OF OTORHINOLARYNGOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI

APRIL 2014

BONAFIDE CERTIFICATE

This is to certify that this dissertation is a bonafide record of work done by **Dr. MANJU JOSEPH** on **"ASSESSMENT OF HEARING LOSS IN HIGH RISK INDIVIDUALS USING HIGH FREQUENCY PURE TONE AUDIOMETRY",** during her M.S. ENT course from 2011 to April at 2014 UIORL, the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. She is appearing for her M.S. Branch-IV Degree examination in April 2014 and her work has been done with partial fulfillment of the regulations of The Tamil Nadu Dr.M.G.R. Medical University, Chennai. I forward this to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation entitled **"ASSESSMENT OF HEARING LOSS IN HIGH RISK INDIVIDUALS USING HIGH FREQUENCY PURE TONE AUDIOMETRY"** is done by me at Madras Medical College, Chennai-3 during October 2012 to October 2013 under the guidance and supervision of **Prof. G. GANANATHAN M.S, DLO.,** to be submitted to The Tamilnadu Dr.M.G.R Medical University towards the partial fulfillment of requirements for the award of M.S DEGREE IN **OTORHINOLARYNGOLOGY** BRANCH-IV

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ACKNOWLEDGEMENT

I owe my great gratitude to **Prof. V.Kanagasabai, M.D., The Dean,** Madras Medical College, for allowing me to undertake this dissertation, **"ASSESSMENT OF HEARING LOSS IN HIGH RISK INDIVIDUALS USING HIGH FREQUENCY PURE TONE AUDIOMETRY"** with much avidity.

I would like to express my deep and sincere gratitude to **Prof. and Director Dr.G.Gananathan, M.S., D.L.O.,** Upgraded Institute of Otorhinolaryngology, for guiding me from the very beginning of the study. His constructive comments and personal guidance have provided a good basis for the present thesis.

I am immensely thankful to **Prof.Dr.R.Muthukumar, M.S., D.L.O., DNB** for the support extended to me. It was from him I learnt how to question thoughts and express ideas.

I am greatly thankful to **Prof. Dr.M.K. Rajasekar, M.S. D.LO.,** Upgraded Institute of Otorhinolaryngology, for his immense support throughout this work.

I express my sincere thanks to respected **Prof.Dr.G.Selvarajan, M.S. D.L.O.,** Upgraded Institute of Otorhinolaryngology. He was always willing to help me and give his best suggestions.

I express my sincere thanks to **Prof.Dr.Rajasekaran, M.S. D.LO.,** Institute of child health, Egmore for his advice and support.

I am greatly indebted to all the Assistant Professors, Upgraded Institute of Otorhinolaryngology, for their thoughtful guidance throughout the work.

I thank The Secretary and The Chairman of The Institutional Ethical Committee, Rajiv Gandhi Government General Hospital and Madras Medical College, Chennai.

I thank the team of audiologists of our department for their help in performing this study.

I express my sincere thanks to **The Traffic Police Commissioner of Chennai,** for his immense support throughout this work.

I would be failing in my duty if I don't place my sincere thanks to those patients who were the subjects of my study.

I would like to express my eternal gratitude to my parents and my family members for their everlasting love and support. Above all I thank The Almighty for his immense blessings.

ABSTRACT

INTRODUCTION

Exposure to loud noise is the most common cause of sensori neural hearing loss in adults. It is an irreversible type of hearing loss typically affecting higher frequency initially then gradually progressing to lower frequency. NIHL is a preventable type of hearing loss through early intervention. Early detection of hearing loss is possible with the help of high frequency pure tone audiometry which may be undetected by a conventional audiometry.

AIMS OF STUDY

- Early detection of Hearing loss in high risk individuals, exposed to noisy environment
- To compare the efficacy of conventional audiometer with the high frequency audiometer in early detection of hearing loss
- Early intervention and prevention of noise induced hearing loss
- To evaluate the relationship between duration of noise exposure and hearing loss
- **METHODOLOGY**

The study was conducted in the department of otorhinolaryngology Government General Hospital Madras Medical College Chennai 60003. A total of 50 subjects were examined within the age group 25 to 55 years .30 people were traffic police and 20 people were drivers in the Central Chennai exposed to nose of an average of 105dB .All the cases subjected to PTA, impedance ,OAE,high frequency pure tone audiometry.

RESULTS

- In my study 83% of subject in the age group 30-40years have high frequency hearing loss and 75% cases above the age 40 years have high frequency hearing loss

-82.5% of cases with normal PTA had high frequency hearing loss

-All the subjects with PTA with dip at 4kHz had high frequency hearing loss

-96.5 %of cases have absence of OAE

-There is considerable relation between duration of exposure and hearing loss and it is proven

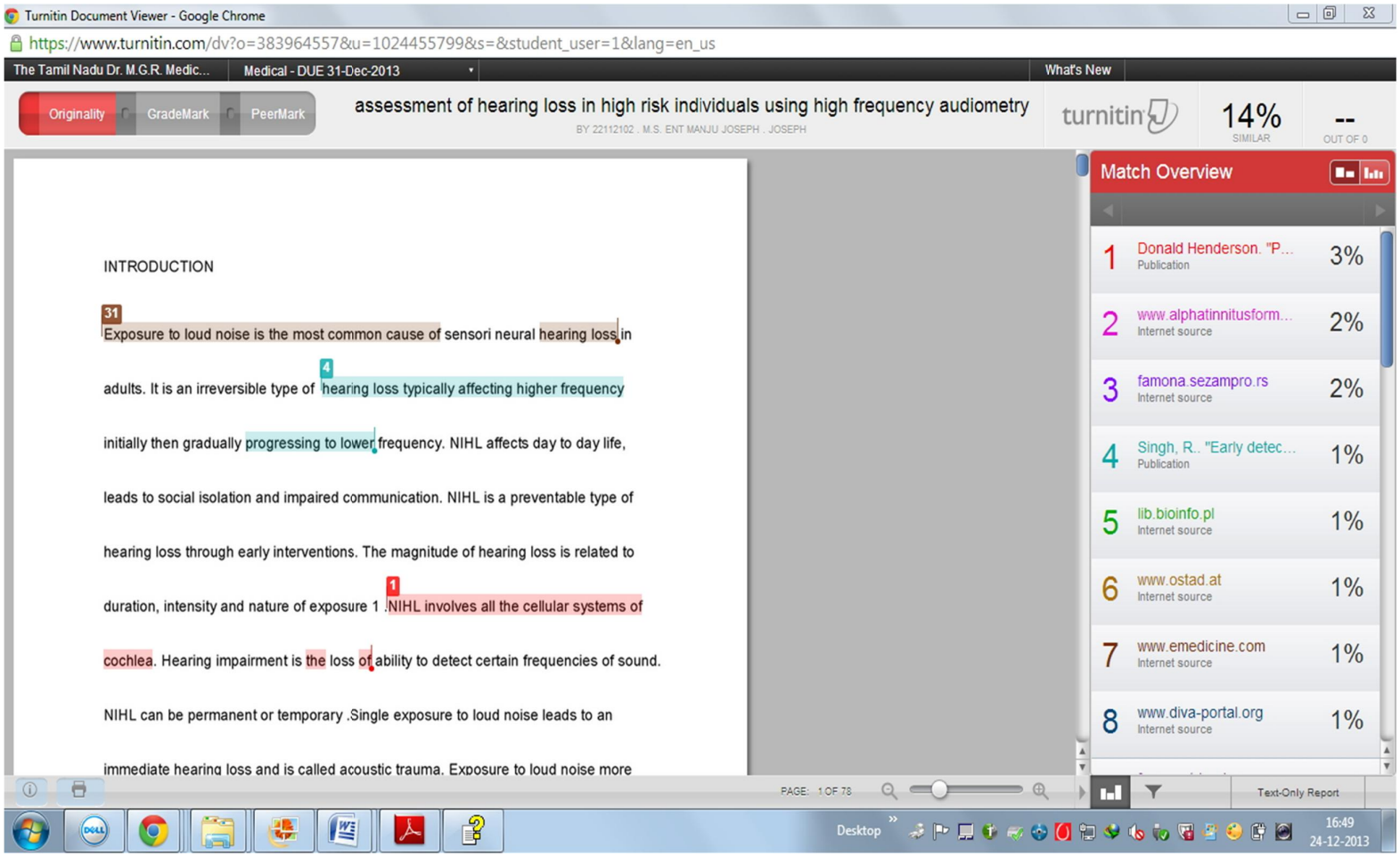
. -All most all cases have symmetrical bilateral hearing loss

COCLUSION

By the time hearing loss is detected using conventional audiometry damage has already been affect the speech frequencies. It will affect the verbal communication of the patient and affect the quality of Life .Therefore by using high frequency audiometry early detection of hearing loss in the high frequency can be detected. It will help us to take an early warning to those working in noisy environment to take preventive measures.

KEY WORDS

High frequency pure tone audiometry, Noise induced hearing loss,conventional audiomery,otoacustic emissions ,high frequency hearing loss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INTRODUCTION
Exposure to loud noise is the most common cause of sensori neural hearing loss in adults. It is an irreversible type of hearing loss typically affecting higher frequency initially then gradually progressing to lower frequency. NIHL affects day to day life, leads to social isolation and impaired communication. NIHL is a preventable type of hearing loss through early interventions. The magnitude of hearing loss is related to duration, intensity and nature of exposure 1 .NIHL involves all the cellular systems of cochlea. Hearing impairment is the loss of ability to detect certain frequencies of sound. NIHL can be permanent or temporary .Single exposure to loud noise leads to an immediate hearing loss and is called acoustic trauma. Exposure to loud noise more
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INTRODUCTION

Exposure to loud noise is the most common cause of sensori neural hearing loss in adults. It is an irreversible type of hearing loss typically affecting higher frequency initially then gradually progressing to lower frequency. NIHL affects day to day life, leads to social isolation and impaired communication. NIHL is a preventable type of hearing loss through early interventions. The magnitude of hearing loss is related to duration, intensity and nature of exposure[1] NIHL involves all the cellular systems of cochlea. Hearing impairment is the loss of ability to detect certain frequencies of sound. NIHL can be permanent or temporary. Single exposure to loud noise leads to an immediate hearing loss and is called acoustic trauma. Exposure to loud noise more than 85dB leads to NIHL. NIHL may be associated with the characteristic 4khz notch. Absence of the notch does not exclude NIHL. Early detection of hearing loss is possible with the help of high frequency pure tone audiometry which may be undetected by a conventional audiometry.

AIMS OF STUDY

- Early detection of Hearing loss in high risk individuals, exposed to noisy environment.
- To compare the efficacy of conventional audiometer with the high frequency audiometer in early detection of hearing loss.
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The degree of hearing loss can range from mild to profound as per WHO grade[2] (table 1)and as per biswas [3] (table 2). The latter is widely used in India.

Table .1

WHO grades of hearing impairment[1]

Grades of impairment	**Audiometric ISO values(average 500,1000,2000,4000Hz)**	**Impairment description**
0(no impairment)	25Db HL	No or very slight hearing problems. Able to hear whispers
1(slight impairment)	26-40dB HL	Able to hear and repeat words spoken in normal voice at 1 metre
2(moderate impairment)	41-60Dbhl	Able to hear and repeat words using raised voice at 1 metre
3(severe impairment)	61-80dBHL	Able to hear some words when shouted into better ear
4(profound impairment)	81dBHL	Unable to hear and understand even shouted voice

Table.2

Widely accepted grading[2]

Audiometric ISO(average of 500,1000,2000Hz)	Grade of Impairment
0 to 25 dB	Normal hearing level for all practical i.e. no deafness. The range between 16and 26dB is termed as very slight deafness by others
26 to 40Db	Mild deafness
41 to 55dB	Moderate deafness
56 to 70dB	Severe deafness
71 to 90 dB	Very severe deafness
Above 90dB	Profound deafness

DESCRIPTIVE ANATOMY OF LABYRINTH

REVIEW OF LITETRATURE

ANATOMY OF LABYRINTH

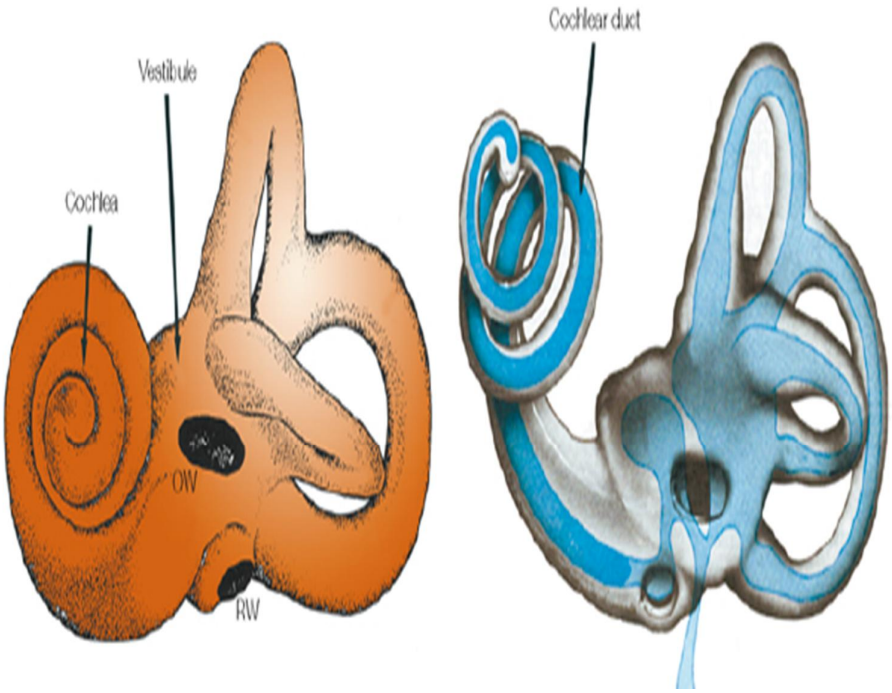
Inner ear lies in the petrous part of temporal bone. Bony Labyrinth consists of cochlea, vestibule, saccule and membranous labyrinth lies within the bony labyrinth. It consists of cochlear duct, utricle, saccule, and three semicircular canals. The space between the inner periosteum of the bony labyrinth and membranous labyrinth is filled with perilymph which is rich in sodium and low in potassium like extracellular fluid. The membranous labyrinth contains endolymph which is rich in potassium and is similar to intracellular fluid .The ionic composition and potentials are essential for the primary function of inner ear because they provide the driving force for mechano transduction. In mammalian cochlea, organ of corti is the receptor organ. It transduces the sound stimuli to electrical signals for transmission to higher auditory pathway .

ULTRA STRUCTURE OF COCHLEA

The name cochlea is derived from Greek literature and means snail. it is the anterior part of labyrinth lying in front of vestibule. It has

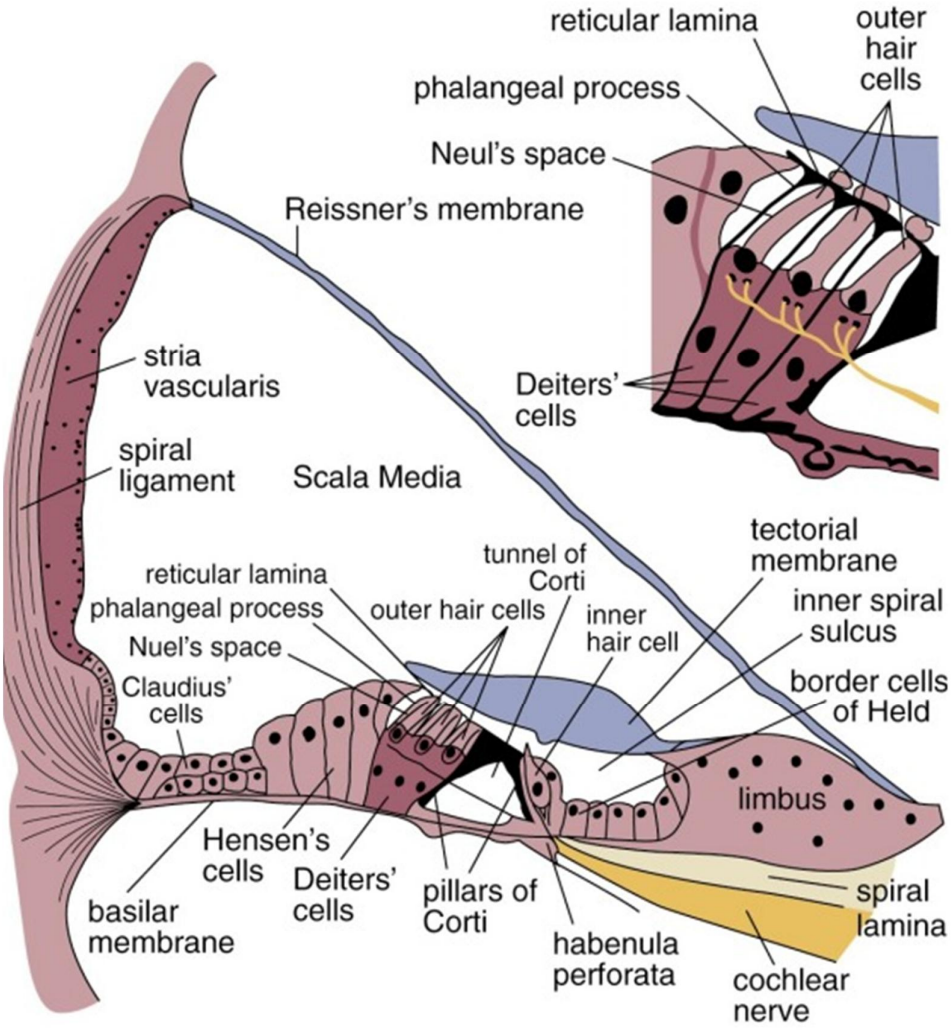
2 ¾ turns and its height is 5mm.The coil of cochlea turns about a central core or modiolous.The base faces the bottom of the internal acoustic meatus, basal coil forms the bulge in the medial wall of middle ear called promontory. Bony spiral lamina arise from the edge of the modiolous and membranous spiral lamina extends from the edge of the bony spiral lamina to outer wall of the cochlea, there by dividing each coil into upper scala vestibule and lower scala tympani. At the apex of the cochlea they communicate through the helicotrema. The middle ear and inner ear communicates through the oval window of the vestibule and stapes foot plate abuts the oval window membrane. Perilymphatic fluid and and subarachanoid space in the posterior fossa communicate through a bony channel called a cochlear aqueduct in the base of osseous spiral lamina. Rosenthal canal is present which accommodate the bipolar ganglion cells of the spiral ganglion. From the Rosenthal canal many tiny canals habernula perforate radiate through the osseous spiral lamina to its rim and carry fascicles of the cochlear nerve to the organ of corti.

BONY AND MEMBRANOUS LABYRINTH



MEMBRANOUS LABRYNTH

Scala Media: It is a spirally arranged tube lying on the upper surface of spiral lamina and its length varies between 29-40mm and it is triangular in cross section. The floor of cochlear duct is formed by bony spiral lamina which separates into two ridges, upper ridge in the spiral limbus from which tectorial membrane originates, lower ridge gives rise to basilar membrane. The sensory organ of hearing, Organ of Corti resides in the membranous labyrinth. Underneath the basilar membrane a layer of spindle shaped cells, tympanic cells, spiral vessels are present. The length of the basilar membrane is 35mm and width increases from base to apex. It separates scala media and scala tympani. Spiral limbus seated on the top of spiral lamina serves as a point of attachment of Reissners membrane and gives rise to tectorial membrane. It is composed of type II collagen fibers. Tectorial membrane lies over the inner and outer hair cells. The border cells of Held lines the inner spiral sulcus.



Cross section of the organ of Corti showing the major cellular structures

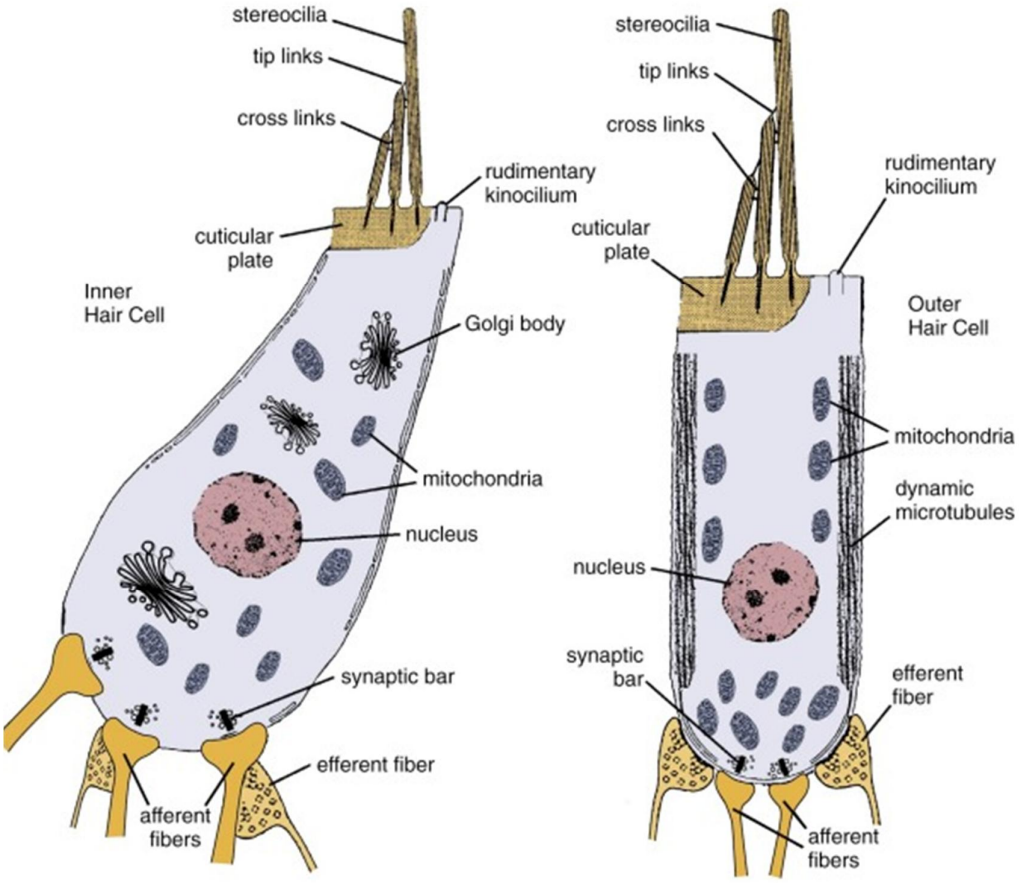
Pillars of corti lie between inner and outer hair cells, which originate from spiral lamina and basilar membrane and converge at the top to form the tunnel of corti. Three rows of outer hairs cells lies lateral to it and supported inferiorly by supporting Deiters cells. Deiters cells has phalangeal process and project apically. Nuels space, a fluid filled space that lies between outer hair cells and phalangeal process of deiters cells. Hensen's cells and Claudius cells lie lateral to outer hair cells. Reticular lamina is formed by phalangeal cells, phalangeal process of deiter's cells and superior surface of hair cells.

Reissner's membrane separates scala media from scala vestibule. It stretches between the bony spiral lamina to the upper part of the lateral wall of cochlear duct. It consists of two layers of cells. Mesothelial layer facing the scala tympani and endothelial layer facing the scala media . The cells within each layer are joined by tight junctions which is impermeable to ions and small molecules.

HAIR CELLS

Hair cells transduce mechanical sound stimulus into the electrical stimuli and stimulate auditory nerve.

Schematic depictions of inner (*left*) and outer (*right*) hair cells



INNER HAIR CELLS

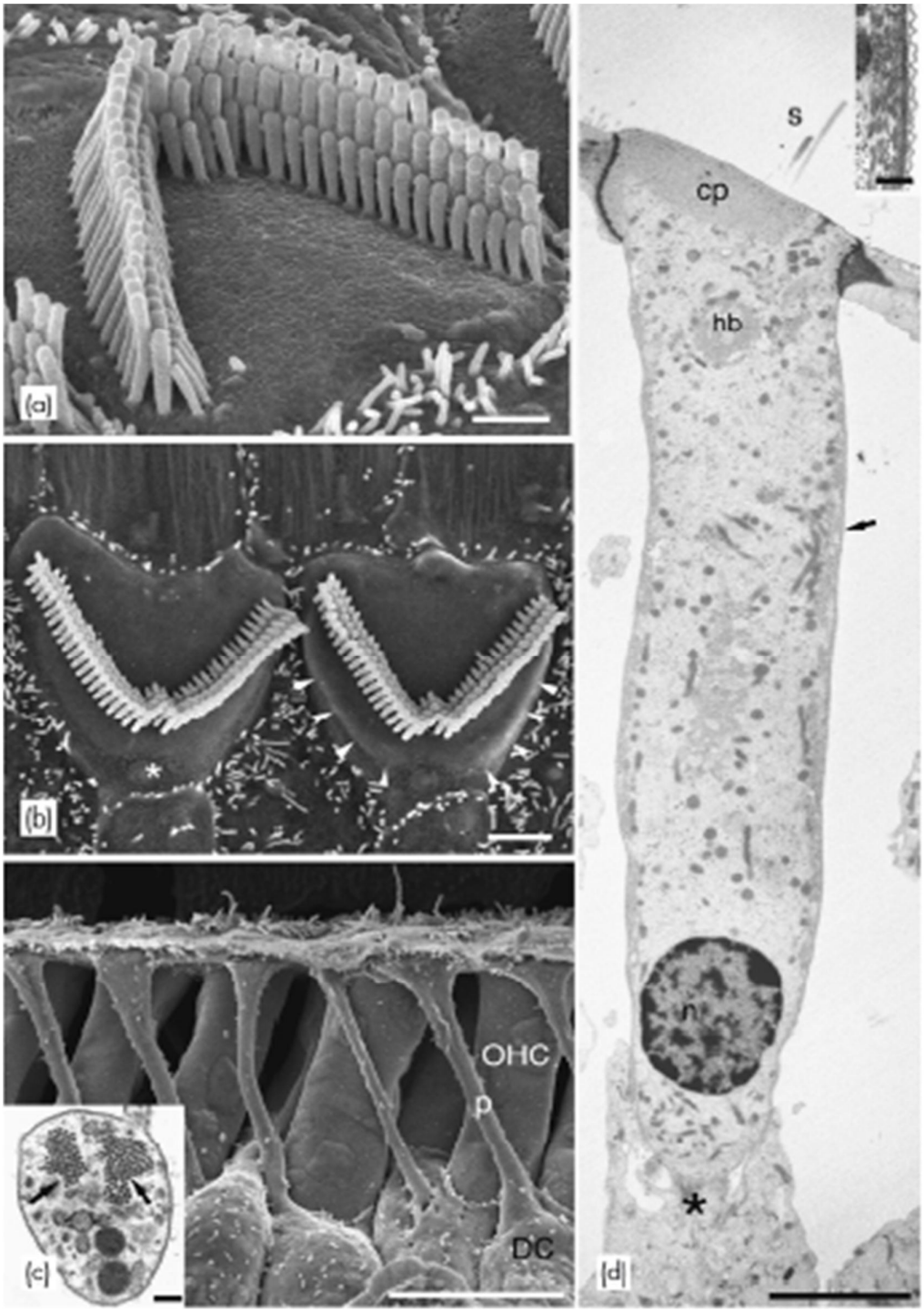
There are 3500 inner hair cells and forms a single row running along the inner side of sensory organ. Pillars of corti which lies between inner and outer hair cells converge to form tunnel of corti. Inner hair cells has flattened or concave apical surface and flask shaped cell body. Actin containing stereocilia are present on inner hair cells. They are arranged according to their height. There are two to three rows of stereocilia in the inner hair cells.

The stereocilia have dense rootlet and penetrate into apical cuticular plate. The stereocilia are connected together by lateral links and bind them both sideways and across the rows. The synaptic pole is at the basal end and afferent fibers make synaptic contact at the basal end. This region contain vesicle, coated and uncoated membranous tubules.

OUTER HAIR CELLS:

The outer hair forms 3 to 5 rows, 12000 in numbers, long cylindrical shaped body. Apex bears several rows of stereocilia. The apical surface is flattened and stereocilia is arranged in a ‘V’ or ‘W’ shaped patterns. The top of the stereociila is in contact with tectorial membrane where the stereocilia may be floating. The Stereocilia are cylindrical and tips are beveled. They contain core of actin filaments, cross linked by plastin and espin proteins. The basal part of the OHC consists of synaptic pole where afferent nerve fibers are connected. The tectorial membrane arises from spiral limbus and extends over the organ of corti and attach close to the Hensen cell.

ELCTRONMICROSCOPIC PICTURE OF OUTER HAIR CELLS



THE LATERAL WALL OF COCHLEAR DUCT.

It consists of three zones, stria vascularis above, spiral prominence below and transitional zone between the two and spiral ligament. Marginal cells facing scala media, intermediate cells and basal cells are the three layers of stria vascularis. Marginal cells have tight junctions connecting highly convoluted membranes of intermediate cells. It helps in maintaining ionic composition of the fluids within the scala media. It contains variety of ion pumps and enzymes.

Tectorial membrane over lays both inner and outer hair cells. It is attached to spiral limbus and loosely connected to supporting cells. The stereocilia of outer hair cells are embedded in the tectorial membrane. It enhances the frequency sensitivity of cochlea .It also contributes to the tonotopic organization of cochlea .They create micro phonics and helps in mechanical amplification.

There are two fluid systems within the cochlea. Perilymph present in between membranous labyrinth and osseous labyrinth and has high concentration of sodium and low concentration of potassium ions and its composition similar to cerebrospinal fluid.

Endolymphatic fluid is present within the membranous labyrinth. It has high concentration of potassium and low concentration of sodium which is maintained by stria vascularis. The Endolymphatic sac communicates with membranous labyrinth via endolymphatic duct and vestibular aqueduct.

SPIRAL GANGLION

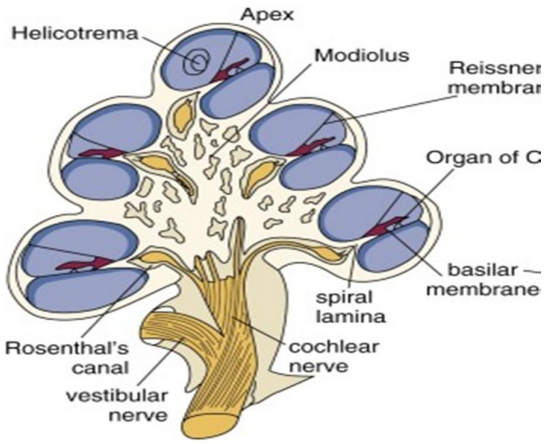
It is located in the Rosenthal Canal. It contains cells bodies of afferent neurons. The dendrites get excited by neurotransmitters released by the organ of corti. There are two types of ganglion neurons namely type I and type II. Type I neurons innervate inner hair cell in a converging pattern while type II neuron innervate outer hair cells in a diverging pattern. The axon of these neurons project to the brain stem. The tonotopic organization of organ of corti is maintained in the afferent system also.

INNERVATIONS OF THE COCHLEA :

Cochlea is innervated by 3 types of fibers that is autonomic, afferent and efferent fibers. Autonomic fibers supply the blood vessels[24] of the cochlea . There are approximately 30000 auditory nerve fibers in

human beings and provide ascending information to central auditory pathway.

Cross section of the cochlea showing the passage of the cochlear nerve through the modiolus to the organ of Corti



The Cochlear nerve transmits auditory information from inner and outer hair cells to the Brain stem. Cell bodies of these afferent fibers forms the spiral ganglion and is located in the Rosenthals canal within the modiolus. The nerve fibers reach the hair cell by passing through the habenulae perforatae. Majority of the fibers are large myelinated fibers and projected from the inner hair cells. 5-10% is unmyelinated fibers which are in contact with outer hair cells. These fibers pass through the modiolous to the spiral canal and project to cochlear nucleus. The cochlear nerve reaches its maximum diameter at the base of spiral canal.

The low frequency fibers occupy the centre and high frequency fibers occupy the periphery of the nerve.

The central process of cochlear nerve reaches the cochlear nucleus, where the initial auditory processing occurs. The cochlear nuclei are divided into dorsal and ventral nuclei. The ventral nuclei are further subdivided into anteroventral cochlear nuclei (AVCN) and posteroventral cochlear nuclei (PVCN). The low frequency fibers are represented ventroloterally while high frequency fibers are represented dorsomedially. The auditory nerve afferents in the AVCN terminate in the principal projection neurons of the cochlear nucleoar complex and expands into very large terminal called bulb of Held. The very low frequency fibers branch to form two end bulbs. These end bulbs contain large number of neurotransmitter vesicles and helps in rapid transmission of signal. AVCN responsible for the original frequency selectivity and sensitivity or cochlear response. The cells in these nucleus analyse the pattern of sound and determine the intensity. PVCN receive input of wide range of frequency and is responsible for the precise time of arrival of sound. The signal is sent to the motor nucleus in the brain stem and midbrain and involved in acoustic startle response. Complex response in the DCN determines what sounds are. All the auditory pathway leaves the cochlear nuclear complex and divides into

dorsal and ventral pathways. The dorsal pathway project directly to inferior colliculus and ventral pathway projects to ipsilateral and contra lateral superior olivary complex. This makes binaural comparison of sound possible. SOC helps in sound localization.

Superior olivary complex contains ‘S’ shaped lateral olivary nucleus, medial olivary nucleus, medial nucleus of the trapezoid body together with smaller periolivary nucleus. Medial olivary nucleus helps in detecting interaural time differences. The ‘S’ shaped nuclei receives an excitatory input from the ipsilateral cochlear nuclei and inhibitory from the contra lateral cochlear nuclei. This helps in detection of difference of sound intensity.

Through the lateral lemniscus the input from the brain stem auditory nuclei is projected to inferior colliculi. The two pathways emerge from the cochlear nuclear complex and join in the inferior colliculus and further analysis is made.

The inferior colliculi contain a central nucleus and outer region composed of dorsal cortex and external lateral cortex. The external portion receives information from cerebral cortex. A tonotopic map is made in the inferior colliculi, by arranging high frequency bands towards the midline of the brain and low frequency bands towards

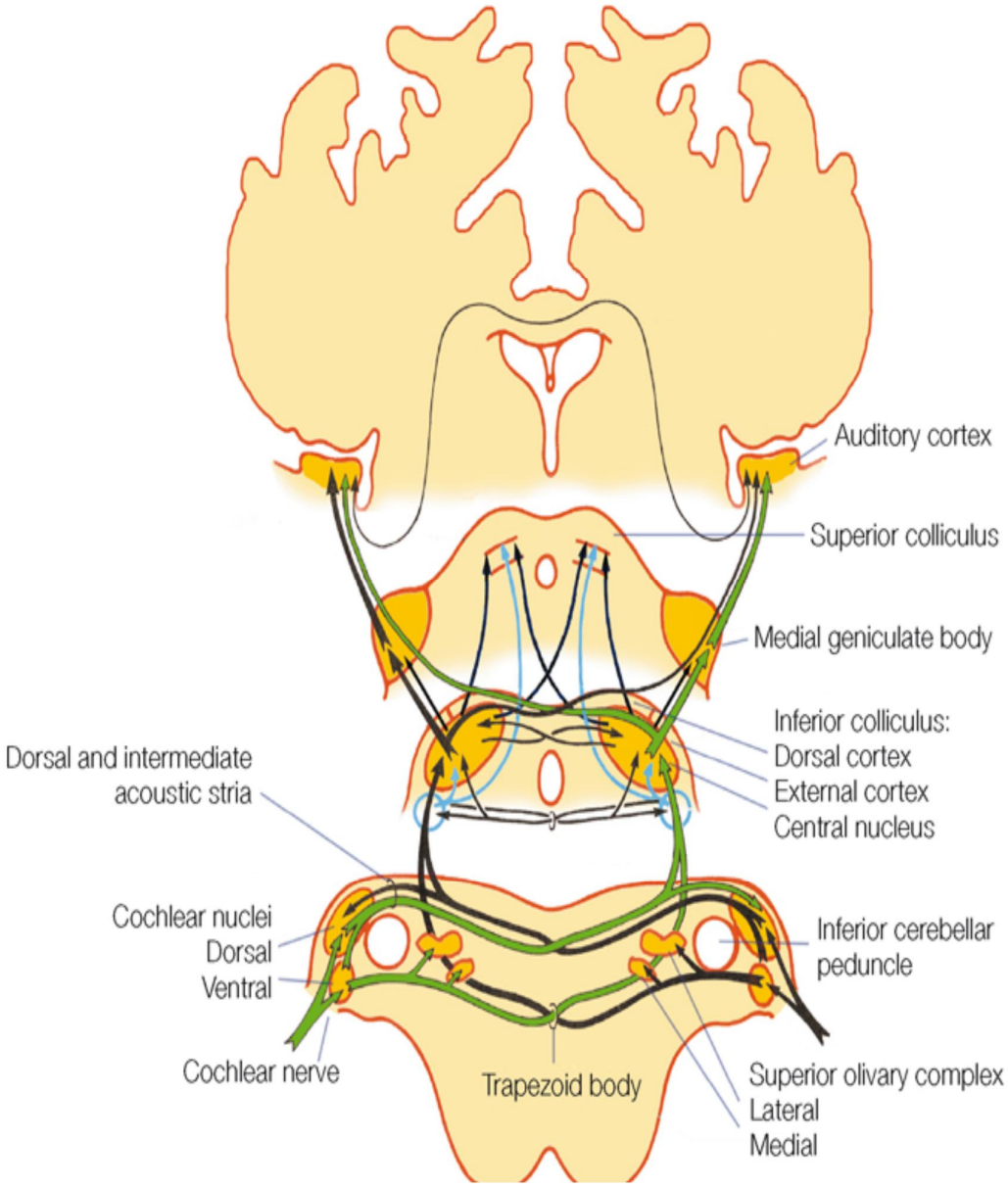
outside. This map is the basis for recognizing patterns in sound and sound localizations. Inferior colliculus also involved in motor response like controlling middle ear muscle, turning head or moving eye in response to sound.

The thalamus receives information from the inferior colliculi.Thalamus has medial geniculate body, posterior nucleus and reticular nucleus, which are involved in auditory function. The ventral division is organized tonotopically into low frequency layers and receives input from the central nucleus of inferior colliculus. The dorsal division is not tonotopically organized and medial division receives multimodal inputs.

From the ventral division of the medial geniculate nucleus, fibers project to Brodmann area 41 within the lateral fissure of the temporal lobe and dorsal division project to non primary areas around A1.

Auditory area also organized into ISO frequency layers, arranged tonotopically, the low frequency sound in the rostral end and high frequency in the caudal end. Most cells in A1 respond to binaural stimulation. The main function of primary auditory cortex is sound localization. Areas around A1 have complex response and it detects specific delays and simultaneous occurrence of harmonically related frequencies.

ASCENDING AUDITORY PATHWAYS



DESCENDING PATHWAYS:

The descending pathway may participate in attention level and anticipation of signals. The major one is olivocochlear feedback loop which originate in SOC and projects back to cochlea. It projects to outer hair cells and is called medial efferent system. It helps in suppression of outer hair cell mobility to make the cell less sensitive and provides protection from loud sounds. The lateral efferent system from lateral superior olivary complex supply inner hair cells which helps in sound localization and binaural comparison.

VASULAR SUPPLY

Labyrinthine artery → common cochlear artery → spiral modiolar artery → radial arteries. Cochlear branch of vestibular cochlear artery supply the spiral ganglion, osseous spiral lamina, limbus, spiral ligament.

VENOUS DRAINAGE:

Apical Region - Anterior spiral vein

Basal region - posterior spiral vein

These two joins with the anterior and posterior division of the vestibular vein in the region of the basal turn of cochlea, to form the vein of the cochlea which empties into jugular bulb.

PHYSIOLOGY OF SOUND TRANSMISSION

Sound is transmitted to the inner ear through the middle ear ossicles, When the sound waves strike the tympanic membrane, it increases tympanic membrane pressure in a frequency sensitive way. An efficient middle ear impedance transformer will change the low pressure high displacement vibration of the sound waves into low displacement and high pressure vibrations. A compression wave is developed in the inner ear fluid due to the vibration of the stapes footplate, which travels across the scala vestibuli, around the helicotrema, and out across the scala tympani toward the round window. An inward motion of the stapes causes an outward motion of the round window. This compression wave travels across the scala vestibule. The pressure in the scala vestibuli is higher than the pressure in the scala tympani. This set up a pressure gradient, which causes the cochlear partition to vibrate .A travelling wave is set up in the basilar membrane. This movement is from base to apex .A shearing motion is developed between reticular lamina and tectorial membrane. This shearing force causes a deflection

of the hair cell stereocilia. This reaches maximum at a particular place of the basilar membrane and decays. Molecular structure at that location of the basilar membrane determines the characteristic frequency. The cochlea is tuned for higher frequency upto 20kHz. This tonotopic gradient is manifested in hair cell height also.

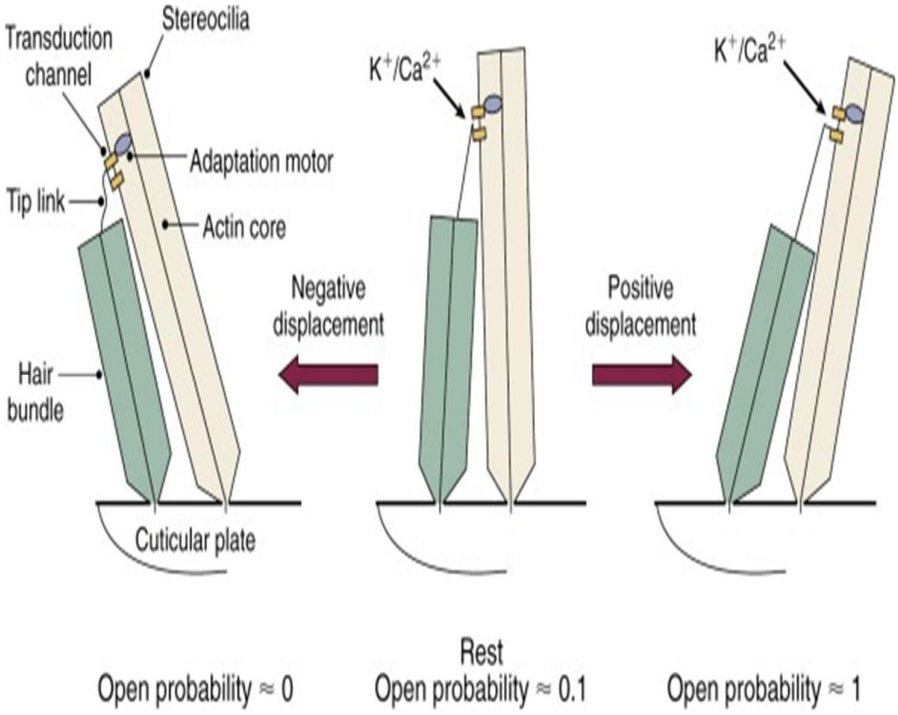
TRANSDUCTION BY HAIR CELLS

The stereocilia on the hair cells are rigid and braced together with cross links, so they move as a stiff bundle. When stereocilia is deflected in the direction of tallest stereocilia, the tip links are stretched and result in the opening of ion channels. Ca^{2+} ions play an important role in the opening of ion channels. The relative motion between tectorial membrane and recticular lamina produce a stimulus which is coupled to stereocilia. This result in opening of ionic channels of stereocilia. Na+, K^{+} and $Ca2^{+}$ will enter the cell. The apical surface which face the endolymph has high positive potential of +80 milli volt and high K^{+} ion concentrations. Inside the cell negative intracellular potential of - 45 milli volt for inner hair cell and - 70mv for outer hair cells is maintained. These potentials combine to give a total of 125mv for inner hair cells and 150 mv for outer hair cells of potential drop across the channels. The K+ ions from the endolymph enters the cell and makes

the cell more positive inside and when channels shut cells become more negative during opposite phase of sound wave. K+ is the main ion involved in transducer mechanism. The main energy comes from the stria vascularis by ion pumping. All these mechanism produce a receptor potential. And neuro transmitters are released from the basolateral membrane of inner hair cell

INNER HAIR CELLS AND ELECTRICAL TRANSDUCTION

The inner hair cells convert mechanical stimulation into electric signals which is transported to brain. This transduction occurs near the tips of steriocilia. Because of shearing motion between adjacent stereocilia, it is transmitted to all hair cells. This leads to opening of the channel protein upon stimulation and leads to depolarization of the cell. This is followed by hyper polarization of the cell where stereocilia are deflected to shorter stereocilia. In these two processes, rapid channel closure and slow adaptation occurs. IHC detects movement of basilar membrane and responds to velocity changes. The basolateral wall of IHC act as a capacitor.



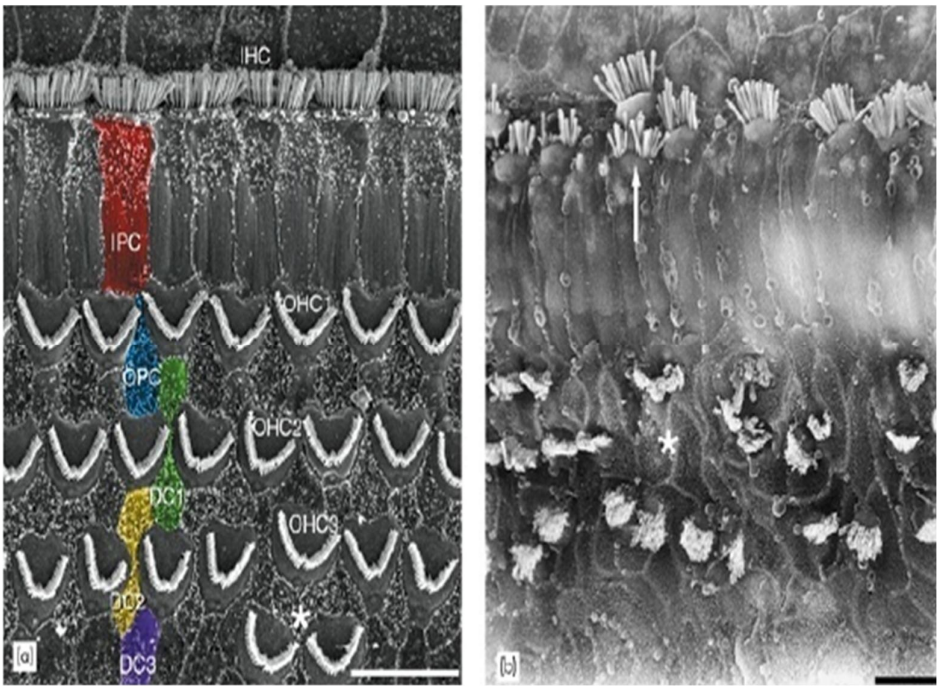
ROLE OF TIP LINKS IN HAIR CELL SIGNAL

TRANSDUCTION

ELECTRICAL RESPONSE OF OUTER HAIR CELL

Outer hair cells are mainly for amplification and sharp tuning of basilar membrane. It is for amplification of sounds at a low pressure level. Outer hair cells act by changing it's length. It will contract upon stimulation which leads to depolarization and pull the basilar membrane. It elongates its length which leads to hyperpolarisation. Prestin is the motor protein in the outer hair cells, responsible for the actions of OHC. On depolarization, the anions dissociate and the surface area decreases leading to contraction. Similarly on hyperpolarisation surface area increases.

ELECTRON MICROSCOPIC PICTURE OF HAIR CELLS



Electron microscopic picture of the upper surface of the organ of corti shows single row of inner hair cells and four rows of outer hair cells, inner and outer pillar cells and deiters cells.

ELECTRICAL RESPONSE OF THE COCHLEA

In response to acoustic stimuli, electric potential can be recorded from the cochlea. Cochlear microphonic potentials represent the mass effects of the transducer currents flowing through outer hair cells. It is an AC response.

SUMMATING POTENTIAL

It may be either positive or negative, depends on the stimulus. It is the distortion component of the outer hair cell response and small contribution from inner hair cells also. It reaches maximum amplitude after the onset of stimulus.

NEURAL POTENTIAL:

It is produced at the onset of stimulus from the massed action potential of auditory nerve.

RESPONSE OF AUDITORY NERVE FIBERS

Action potentials are generated in the auditory nerve fibers, when neurotransmitters are released at the base of inner hair cells. The auditory stimulus is excitatory. The transmitter release and action potential generation is in synchrony with the each cycles of stimulus. When the sound stimulus intensity increases the basilar membrane vibration also increase, mainly its amplitude. This result in activation of inner hair cells and auditory nerve firing rate increases. There is also non linear mechanical response present in cochlea.

PHYSIOLOGY OF NOISE

The term noise is defined as an undesirable sound. It is excessive sound that has potential to harm hearing. It is defined as superfluous, unwanted random sound energy unrelated to sounds being measured, amplified or otherwise studied. Noise is usually a periodic sequence of vibration. Physically, physiologically, psychologically the meaning of noise varies. Physically it is a complex sound having little periodicity that can be measured on its characteristics analyzed. And the physical attributes of noise include frequency, sound pressure, particle velocity, sound intensity, sound energy density etc. Physiologically it is defined as a signal that has no information and intensity varies. Psychologically

it is any sound which is unpleasurable and unwanted. The Psychological attribute includes pitch, loudness, timber intelligibility, annoyance. The physiological measurable attributes include potential to damage hearing.

The frequency of noise is measured in Hertz (Hz), intensity in sound pressure level (SPL) and expressed in decibel (dB). Since it is expressed in decibels it reduces the wide ranging of values to a manageable numbers. Noise may be continuous, fluctuating, intermittent or impulsive[25]. Continuous noise may be relatively constant. Fluctuating noise increases in level over time. Intermittent noise is interrupted for varying time. Impulsive or input noise may be caused by explosions, more common in military environment. It is characterized by short lasting rapidly changing wave fronts and followed by small reverberations and echoes.

The amount of noise, sound pressure level is measured by sound level meter in decibel (dB) using a frequency weighting formula called A-Scale. A-Weighted measurements are preferred in calculating noise exposure. It reduces the sensitivity of the sound level meter in both extreme ranges of audible spectrum.

A standard sound level meter has electronic network and measures noise magnitude automatically in dB. There are four types of

specification for sound level meter type 0, 1, 2, 3. Type 0 sound level meter is used as a laboratory reference standard, type 1 mainly for laboratory and also field where acoustical environment in closely controlled. Type 2 is for general field application and type 3 is for field noise survey application. The frequency range responds for all types from 10Hz to 20000Hz. Sound level meter measures noise according to equal energy principle.

NOISE DOSIMETERS

This a small light and compact instrument worn by workers. It measures the total A-weighted sound energy and express it as a proportion of the maximum A-weighted energy received per day. It is useful whenever the exposure varies during working day. The personal noise dosimeter measures noise dose of percent exposure, experience by the worker. This instrument record on almost any increment of time equivalent level, peak level etc. The dosimeter provides a warning that user is approximately over exposure. The dosimeter works on the basis of equal energy principle.

It states that the hearing damage due to noise exposure is the same whether it arises from a high level noise of short duration or a low

level noise of long duration, provided that the total energy is the same in each case.

NOISE DOSIMETER

NOISE INDUCED HEARINNG LOSS

INTRODUCTION

Noise induced hearing loss is the one of the common causes of permanent hearing impairment. Millions of individuals worldwide have noise-induced hearing loss (NIHL), resulting in a reduced quality of life because of social isolation, and impaired communication with family members, coworkers, and friends.[36] The costs in terms of compensation and early retirement payments for work-related NIHL are immense. The aim of the study is to illustrate the effects of occupational noise exposure on hearing and to improve evaluation helping in early detection of hearing loss in high frequency tones before affecting speech frequency using a high frequency pure tone audiometer. Noise has

obviously a serious impact on hearing and may cause hearing impairment in terms of hearing loss and tinnitus. The working environment is a major factor for noise-induced hearing loss and noise is the source of most prevalent occupational diseases in many countries. Although study about NIHL has been done over a long period, now only the pathomechanism behind it are clearly known. The increments in our knowledge about NIHL helps to improve the detection and prevention of NIHL.

NATURE OF HEARING LOSS

Noise is an undesirable sound. Noise may be continuous, fluctuating, intermittent or impulsive. Continuous means a constant steady noise. Fluctuating means noise varies over time, while intermittent noise are interrupted over time periods

Depending on the time period of exposure, hearing loss may be temporary or permanent. Temporary hearing loss is a reversible loss i.e. temporary threshold shift. It may be accompanied by tinnitus, dysplacusis. Recovery may range from minute to hours. Magnitude of TTS depends on the intensity, frequency and temporal pattern of noise. High frequency sounds are more dangerous than low frequency sounds. If the ear is re-exposed to loud noise before recovery, permanent

Threshold Shift(PTS) will occur. PTS is due to the structural damage of cellular system of cochlea. Repeated episode of this TTS leads to PTS. In PTS focal loss of hair cells and degeneration of nerve fibers[37] in acoustic trauma that is a single short lasting exposure of sound. NIHL occurs due to continuous chronic exposure of sound. Occupational noise is always sensorineural and affects mainly OHC. Typically the threshold shift occurs bilaterally.

It produces characteristic notching at 4 kHz with high frequency hearing loss. NIHL produce maximum 75dB HL at high frequencies and 40dB at low frequencies. Hearing loss is initially at higher frequency. Once noise exposure is discontinued hearing loss does not progress. The development of hearing loss mainly starts in the high frequency level and gradually progress to middle and low frequencies. Regular exposure to low intense noise also increases the hearing threshold. Asymmetric pattern of hearing loss is also noticed in some persons involved in sports like shooting etc.

PATTERN AND MECHANISM OF NOISE INDUCED COCHLEAR PATHOLOGY

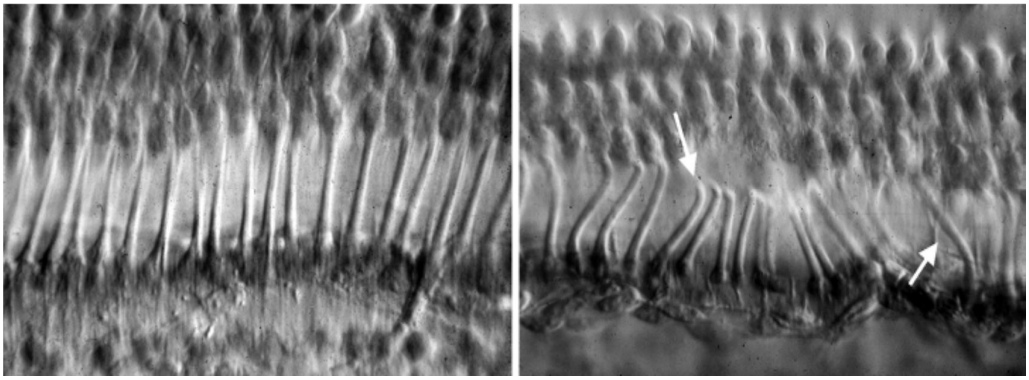
Cochlea is a highly energy consumptive biological system. Each cellular system of cochlea is vulnerable to noise exposure. Basilar membrane is 200 times stiffer at the base[3]. It results in an impedance gradient from apex to base. So high frequency creates a maximum vibration at base and low frequency at the apex. The damage to pillar cells leads to abrupt change in the basilar membrane impedance gradient. It may lead to cell death.

SENSORY CELLS

Outer hair cells are most vulnerable to noise exposure. Noise exposure distort stereocilia of OHCs. Brownell[4] showed that OHC movement is due to the synchronized depolarization of stereocilia .With complete loss of OHC 40 to 50 db HL can occur. The K+ ions are circulated out of hair cells through the supporting cells namely Claudius cells and Henson cells. Eventually the fibrocytes in spiral ligament transfer the potassium ions into stria vascularis. Type II fibrocytes[5] are the most vulnerable to noise exposure and its loss closely corresponds to spectrum of noise exposure. The stereocilia may be shortened or break on noise exposure.

IHC are more resistant to noise exposure. When complete loss of OHC in a region leads to loss of IHC and VIII nerve fibers. Temporary effect of noise can be seen in IHC and VIII nerve [6]

Because of high rate of synaptic activity VIIIth nerve fibers swells up due to intense noise exposure. Failure to recycle glutamate accumulated in the dendrite terminal cause excitotoxic effect leads to swelling of postsynaptic cell bodies and dendrites [7]



Phase-contrast views of outer pillar cells. Left panel shows normal anatomy.Right panel is after exposure to 50–155 dB SPL impulses. Notice (arrows) the detachment at the level of the cuticular plate.

COCHLEAR VASCULAR SYSTEM

The vascular system of cochlea plays a complicated role in NIHL. The Cochlear blood flow depends on the systemic and local changes

within the cochlea. Systemic changes is influenced by the sympathetic[8] influence and local auto regulation[9]. Sympathetic influence of cochlea is coming from the innervation of stellate ganglion and unilateral superior cervical ganglion. The response of the vascular system depends on the type of noise and duration of exposure[10]. With continuous noise exposure there will be an initial increase, followed by a decrease in cochlear blood flow. The interruption of the sympathetic innervations of cochlea reduces its susceptibility to noise. On exposure to loud noise, sensory cells detach from the organ of corti and result in a cleft between the 1st and 2nd rows of OHC. The endolymph enters the cleft and produces an osmotic and ionic changes which results in cell death. A combination of impact noise and continuous noise is more harmful to the ear.

DYNAMICS OF COCHLEAR PATHOLOGY.

After an exposure to loud noise, the cochlear pathology especially hair cell loss continues even for 30 days[12]. The lesion continues to expand in the basal direction and cells die by both apoptosis and necrosis. A study on Chinchillas that were exposed to loud noise for 24 hours over a period of 6 months showed that they developed a stable level of threshold shift which remained so for the 6 months study period. This is designated as Asymptomatic threshold shift[13]. Then

ATS grew at a rate 1.7 db HL for each dB increase in noise level. Henderson and colleague showed that low level impact (95-115 db) produce HL of 1.9 dB and above 120dB, an increase of 3-5dB was recorded. When a peak level of noise exposure exceeds a critical level, the direct mechanical failure occurs and critical level varies with species.

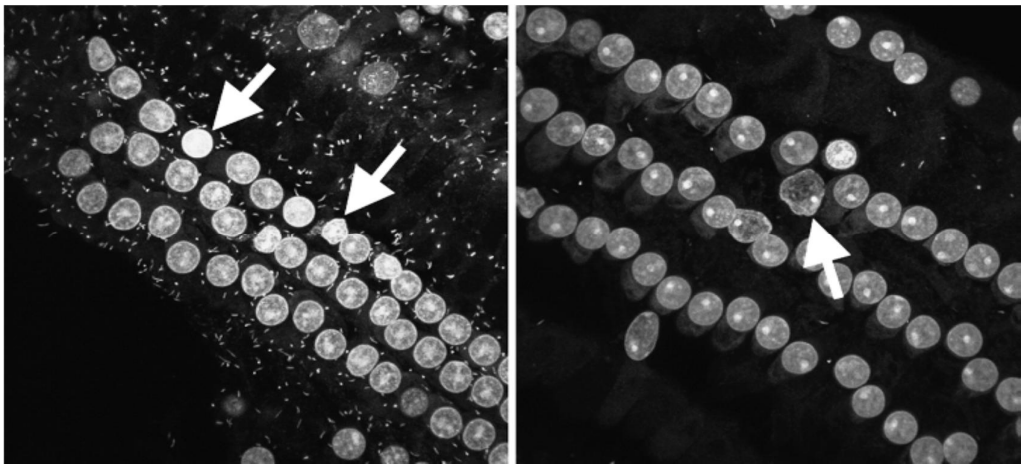
NOISE AS A STRESSOR TO THE COCHLEA.

Cochlea function at a high metabolic rate and the main energy source is stria vascularis. It contains large number of mitochondria which maintain ionicity and polarity of endolymph by extruding k^+ constantly[14]. The reactive oxygen species (ROS) in the cochlea are produced by mitochondria. Mitochondria consumes 98% of Oxygen and converts ADP to ATP. 1-2% O2 is converted to superoxide. Because of high level noise exposure high level of ROS[16] is generated. These are two factors for high level of ROS generation. First cochlear metabolism increase at a faster rate. Secondarily cochlear blood flow decreases, ischemia develops leading to shortage of O2 for mitochondrial functions with resultant superoxide production. When reperfusion occurs blood flow increases providing a rich O2 supply leading to a burst of superoxide generation[17]. Finally cells are destroyed with extrusion of cellular contents into extracellular matrix. Reactivity continues for

several days due to which hair cells continue to die after exposure to loud noise.

PATHWAYS OF SENORY CELL DEATH

Noise exposure produces both apoptosis and necrotic cell death[18]. Following noise exposure the level of phosphates, calcineurin and Bcl-xl/Bcl-2 associated death promoter level increases. There is a short latency before apoptosis starts following noise exposure and once started apoptosis continues. It may be converted to necrosis because of lack of energy to finish apoptosis. The apoptosis starts from the centre to basal end of cochlea and is driven by lipid peroxidation[19]. Impulse noise produce damage to OHC by ROS production. OHC are shortened and their nuclei migrate from basal pole to middle of the cell and finally it shrinks.



Examples of apoptotic cell death (left) and necrotic cell death (right).

COCHLEAR RESPONSE TO STRESS FROM NOISE

Cochlea has several defense mechanisms to protect it from high level noise. It produces heat shock proteins[20]. It also increases the activities of antioxidant system namely glutathione reductase, catalase, gamma glutamyl cysteine synthatase. .

In short, noise damage cochlea and causes hearing loss up to 50dB. OHC are more vulnerable to noise exposure. Noise damage cellular system of cochlea by production of ROS which in turn initiates death by apoptosis and necrosis and this continues for few days after noise exposure.

PREDISPOSING FACTORS

Some patients are more prone to noise induced hearing loss suggesting a genetic basis for it i.e. physical characteristics and cochlear ultra structure, ahl gene[26] have been proposed as contributing to susceptibility.

- smoking[27]
- Previous damage to cochlear[28]
- hypercholesterolemia[29]

- Hypertension[30]
- Recreational drug users
- Ototoxic drug exposure
- Single nucleotide polymorphism of catalase gene involved in oxidative stress are involved in noise susceptibility

CLINICAL FEATURES

The middle aged male people with complaints of tinnitus with or without hyperaccusis

In the early stage patients present with a history of hearing difficulty in the presence of background noise. They usually describe lack of clarity to speech.

Tinnitus is an early symptom, especially post exposure tinnitus. Hyperaccusis may be found in 40% of noise exposed individuals. With further progression of disease, patients may complain of obvious hearing loss. Noise induced hearing loss is more common in men and it leads to social isolation. There may be embarrassment, loss of confidence, anxiety, and frank depression.

OTHER ADVERSE EFFECTS BY NOISE EXPOSURE

Vestibular Dysfunctions[31] can be associated with noised induced hearing loss, due to common arterial supply to cochlea and vestibular end organ and similarity in the ultra structure of vestibular and cochlear cells. Sometimes asymptomatic vestibulopathy can progress to severe disturbing vertigo under certain situation[32].

Non auditory complication is impairment of general health[33] due to prolonged activation of autonomic nervous system and pituitary adrenal complex.

It also affects gastro intestinal motility and can cause peptic ulcer[34]. Sometimes circulatory problems and hypertensions can occur in noise induced hearing loss.

Exposure to loud noise can affect task performance and cause emotional upset.

INVESTIGATIONS

Audiometric testing is the only diagnostic evaluation of NIHL.

Pure tone audiometry - The classical pattern of high frequency hearing loss with notch at 4/6 kHz

HIGH FREQUENCY PURE TONE AUDIOMETRY

High frequency air conduction testing is done from 8000 to 20000 kHz. Routine PTA covers 125 to 8 kHz, although Human cochlea responds up to 24 kHz. Because most sounds occurring in our day to day life fall within 125 to 8 kHz. Secondly normative threshold data below 8 kHz are less affected by acoustic factors.

The high frequency pure tone is not used routinely due to the acoustic characteristics of the high frequency pure tone i.e. it covers only 1.5 octaves whereas conventional audiometry covers 6 octaves. The wavelength of high frequency tones are short, makes transducer mental coupling difficult. The standing waves may not allow true thresholds to be assessed. They may also cause threshold variation in the subject. The calibration of audiometer is found to be difficult. Different studies show that high frequency audiometry is valuable in early diagnosis of the traumatic effects of high intensity noise, ototoxic drugs, dietary factors[35].

When one-fourth wavelength of incident sound same as ear canal length was used, resonance and anti resonance developed at the tympanic membrane which resulted in inter subject variability. This

problem is now solved with using special probe microphone to measure the sound pressure at the level of drum.

Now special head phones are used HDA 200 sennheiser head phones. They are closed dynamic headphones specially designed for extended high frequency testing. They have excellent passive attenuation, very high quality sound reproduction with single side cable.

They have added headband with adjustable cushion, circum aural ear pads. Using this type headphones measurement is done with steady state sine wave signals, it has very high passive sound attenuation.

HDA 200 AUDIOMETRIC HEAD PHONE

HIGH FREQUENCY PURE TONE AUDIOMETRY

TYMPANOMETRY - To Confirm Normal Middle Ear Functions

Cortically evoked response audiometry is the most valuable objective test because of the following reasons.

It has good frequency specificity

It is non invasive

It is recorded from higher auditory level.

OAE

Helps to detection of OHC damage

MRI - to evaluate vestibular sahawanomma

Tinnitus pitch and intensity matching can be done.

DIFFERENTIAL DIAGNOSIS

Inner ear autoimmune disease

Inner ear genetic sensorineural hearing loss

Inner ear ototoxicity

Inner ear presbycusis

Sudden sensorineural hearing loss.

DIAGNOSIS

History of prolonged unprotected exposure to loud noise and no history of other otological problems.

Audiogram shows High frequency hearing loss

The effect of ageing can be avoided using NPL tables and black book.

MANAGEMENT

No well recognized and validated treatment is specifically available.

PREVENTION

Health and safety Act 1974 minimizes the risk to Employees. The 1989Noise Act work Regulation describes two action

Levels for daily noise exposure - first action level at 85 dB (A) and a Second at 90dB (A). In addition there was a peak action level of 140 dB.

These have recently been replaced (April 6, 2006) by The Control of Noise at Work Regulations, 2005, where each action level is 5 dB lower respectively. The employees are educated and provided appropriate hearing protection. The use of this hearing protection is at the discretion of the employee until the second or peak action when it becomes compulsory.

Reduction of noise level at the source of production

Specific protection of the individuals who are at risk

Ear plugs - Approximately will provide protection 10to 15 dB of sound protection

Ear muffs- Provides at least 15to 30 dB sound protection

Health education about NIHL and preventive aspects

Both ear plugs and ear muffs should be worn in areas of loud noise exposure. These hearing protectors should be worn all the time of exposure. Even if they remove for 15mts in an eight hour work period, the efficacy is reduced to half.

Early detection - periodical audiological check up

Active noise reduction with electronic method of sound attenuation

Pharmacological Protection

Hypoxia is the major cause of NIHL. Based on the fact that oxidative stress is the major cause of Nose induced cochlear damage, pharmacological strategies have been developed to protect cochlea against this[2]. NIHL leads to mitochondrial dysfunction, glutamate

induced excitotoxity and depletion of GSH. Based on this Acetyl L. Carnitine helps to maintain mitochondrial biosynthesis. Also carbamathione, a glutamate antagonist, GSH repletion agents D-methionine all improved hearing. Antioxidants also play on important role in noise exposed persons.

RECOMMENDATIONS FOR PREVENTION OF NOISE INDUCED HEARING LOSS BY THE WORLD HEALTH ORGANIZATION

The World Health Organizations Programme is concerned with prevention of Deafness and Hearing Impairment especially in developing countries. It also promotes strategies for the prevention of the major causes of hearing impairment and deafness which constitute public health problems. Prevention and management of Noise Induced Hearing loss should be started in primary health care and it should be appropriate, adequate and affordable.

Hearing loss can be attributed to noise by the following criteria

History of noise exposure - 40 hours per week equivalent continuous noise level for a 50 year life time. Audiometric criteria include predominantly bilateral sensorineural HL.

Tympanometry should be normal

By creating awareness about the adverse effects of noise exposure through school and all health educational programme and campaigns. Also create awareness about the importance of hearing in day to day life.

National programme - It is a multisectorial integrated approach .All the programme can be implemented through the PHC. It has the influence on local community.

Training - We have to train the persons to carryout noise surveys and audiometric testing.

Occupational Noise: It is a major problem for hearing loss in developing countries. Through programme, training the workers, education, and audiology testing and usage of protectors, we can control it.

Rapid urbanization of developing countries result in high level of traffic noise. This has to be reduced by devising regulations, proper use of silencers, effective land use planning.

Socioeconomic Impact - Data should be collected to enable determination of social and economic consequences of NIHL.

We have to limit the high noise leisure pursuits Effective collaboration with NGO and WHO and its members Epidemiological data should be collected about the prevalence of NIHL Data should be for the better understand the development and progression of NIHL.

-Screening methods should be developed for early identification and intervention of NIHL.

Epidemiological Data.

There is a serious shortage of accurate epidemiological information relating to NIHL especially in developing countries. It is recommended that

- Representative surveys should be conducted for the prevalence of significant NIHL in less developed countries.

- High quality longitudinal data is gathered to better understand the development and progression of NIHL.

- Effective screening methods be developed for early identification of and intervention against NIHL.

Research Priorities. There is considerable ignorance about the pathogenic mechanisms of noise-induced hearing loss and effective

means for its prevention. It is recommended that priority should be given to research on the following subjects

- Mechanical, metabolic and molecular mechanisms of NIHL;

- Investigation of low cost medications for prevention;

- Engineering research on technical measures for noise abatement and improving hearing protectors;

- Studies on the risk factors for NIHL including individual susceptibility to noise damage.

- Studies on the interaction of other toxic agents with noise

Assessing Noise Risk

Noise level 80dB or more for many years produce hearing impairment and noise 115 dB SPL produce permanent damage. The damage risk criteria depend on the intensity and noise duration.

Risk estimation of noise exposure is a complex process as noise exposure to leisure activities also affected hearing mechanism. Now exposure to noise with CD/MP3 walkman is also an important source hearing loss[23]. According to OSHA exposure steady state equal to 90dB over I8 hour work period is allowed. If the sound exposure is <90dB

more period is allowed and if it is greater than 90 dB this exposure time should be reduced.

For every 3 dB increase in SPL, working period should be reduced to half. According to OSHA every 5dB rise in noise level the exposure has to be reduced to 50%. The people with noise exposure during non work time should be careful as it may interact with work exposure and increase hearing loss.

Occupation safety health regulations

According to OSHA 80dB 8 hour work time in permitted. There are 5 regulations according to OSHA.

1) Identification and exposure leads.

2) Protection of workers from hazardous exposure.

3) Hearing test annually.

4) Workers trained annually& recorded.

MATERIALS AND METHODS

AIMS AND OBJECTIVES:

- Early detection of Hearing loss in high risk individuals, exposed to noisy environment
- To compare the efficacy of conventional audiometer with the high frequency audiometer in early detection of hearing loss
- Early intervention and prevention of noise induced hearing loss
- To evaluate the relationship between duration of noise exposure and hearing loss

STUDY PLACE: Government General Hospital, RGGGH Chennai

Collaborative department: Upgraded Institute of Otorhinolaryngology :

Department of audiology

Study design : Prospective

Study period : October 2012 to October 2013

INCLUSION CRITERIA

- Age : Above 20 years
- Sex : Both Males & Females
- Individuals exposed to noisy environment
- Middle ear function should be normal

EXCLUSION CRITERIA

- Age below 20 years
- External or middle ear abnormalities
- Individual with conductive hearing loss
- Actively discharging ear
- Individual with prior history of ear surgery.
- People with family history of HOH

INVESTIGATIONS

(1) Otoscopic Examination

(2) Pure tone audiometry

(3) Examination under Microscope

(4) Impedance audiogram

(5) OAE

(6) High frequency pure tone audiogram

METHODOLOGY

The study was conducted in the department of otorhinolaryngology Government General Hospital Madras Medical College Chennai 60003. A total of 50 subjects were examined within the age group 25 to 55 years .30 people were traffic police and 20 people were drivers in the Central Chennai exposed to noise of an average of 105dB . All of them working for more than 2 Years.

Relevant history noted. All persons were first examined by Otoscopy and under microscope. and ruled out any middle ear and external ear pathology. All cases subjected to pure tone audiometry. From 125 to 8 kHz's the response were based on subject activation of Hand held response buttons. Steps of 5dB were used in obtaining results. The results were expressed in dB HL. Also bone conduction from 250 to 4 kHz.

According to the PTA finding, cases with conductive hearing loss were excluded from the study. Then the cases were divided into NL PTA, PTA with dip at 4 KHz. All these cases were subjected to tympanometry. All the patients with normal tympanogram and presence of acoustic reflex were included in the study. All the included subjects were subjected to otoacoustic emission study and noted

whether it was absent or present. Only few cases show presence of otoacoustic emission. All the cases were subjected to high frequency pure tone audiometer air conduction test from 8 KHz to 16 KHz. The response is measured using a hand held button .The HFA have a very special head phone with HDA 200 Audiometric headphone. Closed dynamic Headphone with excellent passive ambient sound attenuation. The response were noted in both ear.

MADRAS MEDICAL COLLEGE & RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL

INSTITUTE FOR REHABILITATION OF SPEECH & HEARING HANDICAPPED

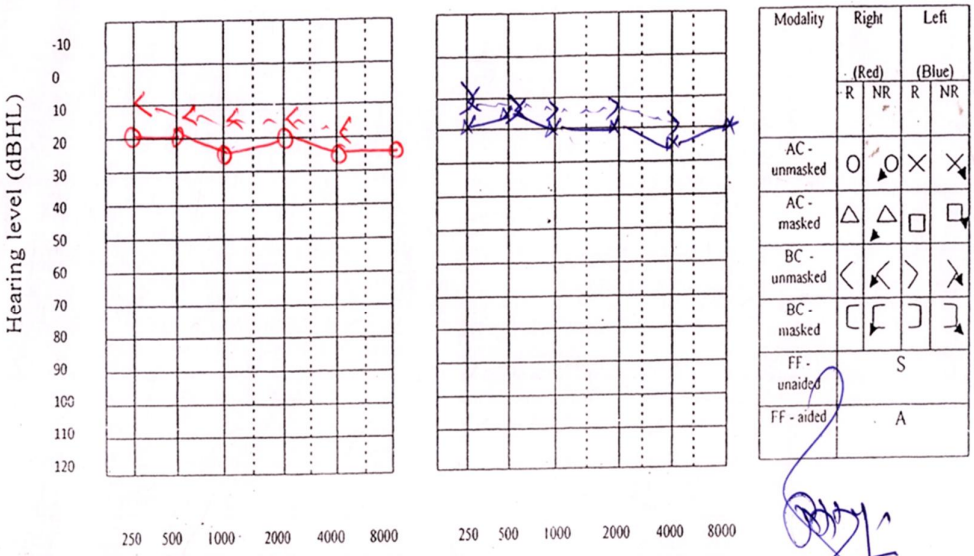
CHENNAI-03

PURETONE & SPEECH AUDIOMETRY

Name: Gandhi Age / sex: 45 M Date: 8/7/13

O.P/I.P No: 10124 Instruments used: Reliability:

Right ear Left ear



Modality	Right (Red)		Left (Blue)	
	R	NR	R	NR
AC - unmasked	O	O	X	X
AC - masked	△	△	□	□
BC - unmasked	〈	〈	〉	〉
BC - masked	[	[	]	]
FF - unaided	S			
FF - aided	A			

Ear	PTA (dB HL)	SRT (dB HL)	SIS (%)	SDT (dB HL)	UCL (dB HL)
Right ear					
Left ear					

Provisional diagnosis: Right ear – Bilateral normal hg.
Left ear -

Recommendations/ Remarks

Student clinician

Audiologist

MADRAS MEDICAL COLLEGE & RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL

INSTITUTE FOR REHABILITATION OF SPEECH & HEARING HANDICAPPED

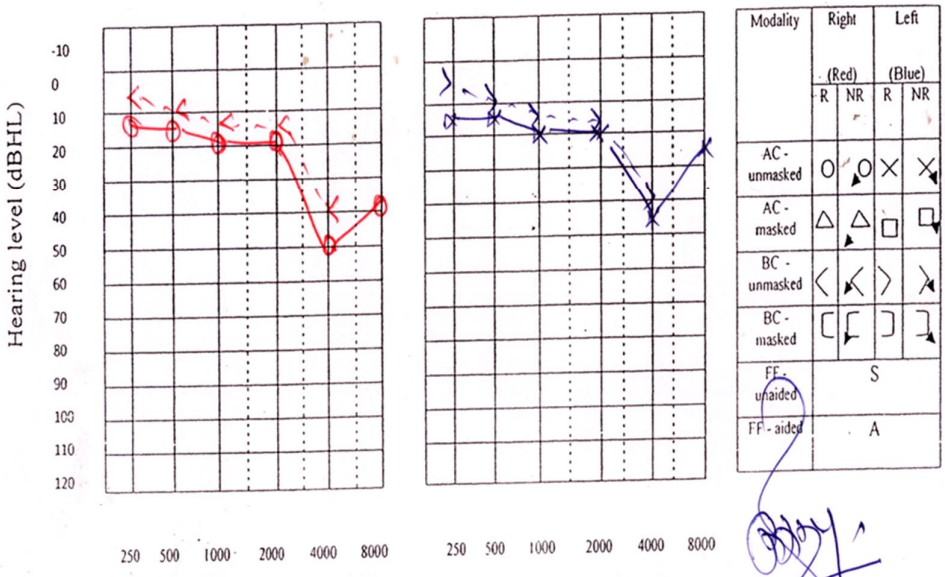
CHENNAI-03

PURETONE & SPEECH AUDIOMETRY

Name: R. Raja Age / sex: 31/M Date: 8/7/13

O.P/I.P No: 7324 Instruments used: Reliability:

Right ear Left ear



Modality	Right (Red)		Left (Blue)	
	R	NR	R	NR
AC - unmasked	O	O	X	X
AC - masked	△	△	□	□
BC - unmasked	〈	〈	〉	〉
BC - masked	[	[	]	]
FF - unaided	S			
FF - aided	A			

Ear	PTA (dB HL)	SRT (dB HL)	SIS (%)	SDT (dB HL)	UCL (dB HL)
Right ear					
Left ear					

Provisional diagnosis: Right ear –
Left ear -

} Bilateral WNHL c 4kHz notch

Recommendations/ Remarks

Student clinician

Audiologist

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INSTITUTE FOR REHABILITATION OF SPEECH & HEARING HANDICAPPED

CHENNAI-03

IMPEDANCE AUDIOMETRY

Name: Gandhi Age / sex: 45/M Date:-

O.P/I.P No: 10124 Instruments used:

Tympanometry:

Ear	Tympanogram type	Peak pressure (dapa)	Static compliance (ml)	Gradient (dapa)	Ear canal volume (ml)
Right ear	'A'	-15	0.8	45	1.1
Left ear	'A'	-5	0.5	40	1.3

Acoustic Reflex thresholds:

Frequency	Right ear		Left ear	
	Ipsilateral	Contralateral	Ipsilateral	Contralateral
500 Hz				
1000 Hz		Present		
2000 Hz				
4000 Hz				

Reflex Decay test:

Ear	500Hz	1000Hz
Right ear		
Left ear		

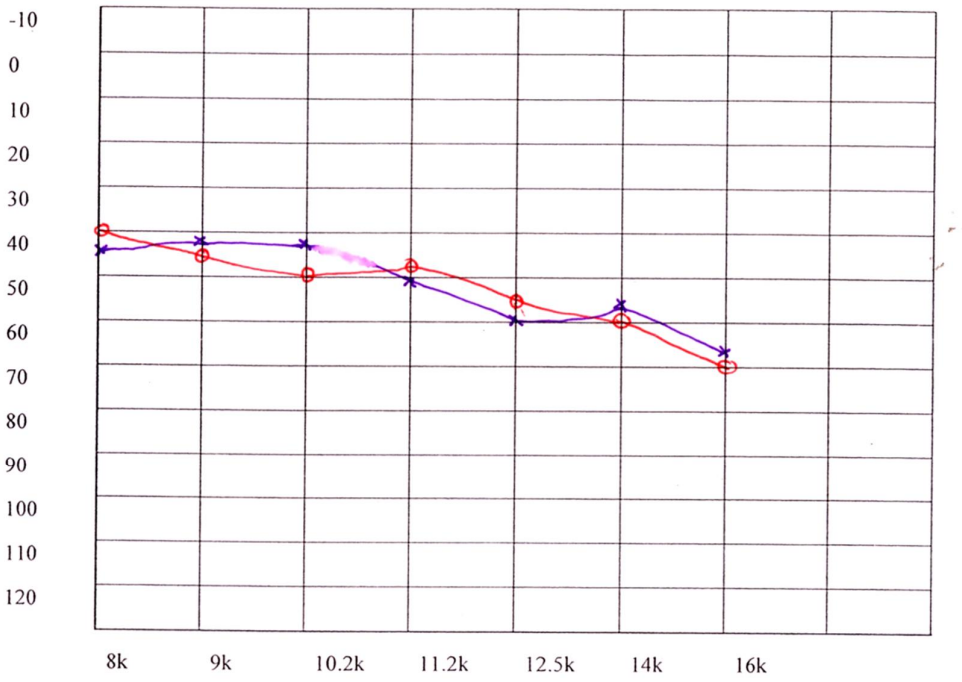
Impression: Suggestive of no middle ear pathology in both the ears.

Recommendations:

Student clinician **Audiologist**

Name : Gandhi Age: 45 Sex : m OP No. : 10124

HIGH FREQUENCY PURE TONE AUDIOGRAM



IMPRESSION :

RESULTS AND ANALYSIS

The following data were obtained from the study conducted at RGGGH Chennai in noise exposed individuals of Chennai central zone.

The following results and analysis were concluded from my study.

The following parameters were measured-

Unilateral or bilateral noise induced hearing loss

Duration of work

Age distribution

Normal PTA with high frequency hearing loss

Otoaccoustic emissions

(If P-Value <0.05 then statistically significant)

The Normality tests Kolmogorov - Smirnov and Shapiro-Wilks tests results reveal that the variables do NOT follows Normal distribution. Therefore to analyse the data Non-parametric methods are applied. To compare the between two groups Mann-Whitney U test is applied. To compare three or more groups Kruskal Wallis test is used. Correlation is used to calculate the degree of relation between variables. To analyse the data SPSS version 20.0 is used

Frequency Tables.

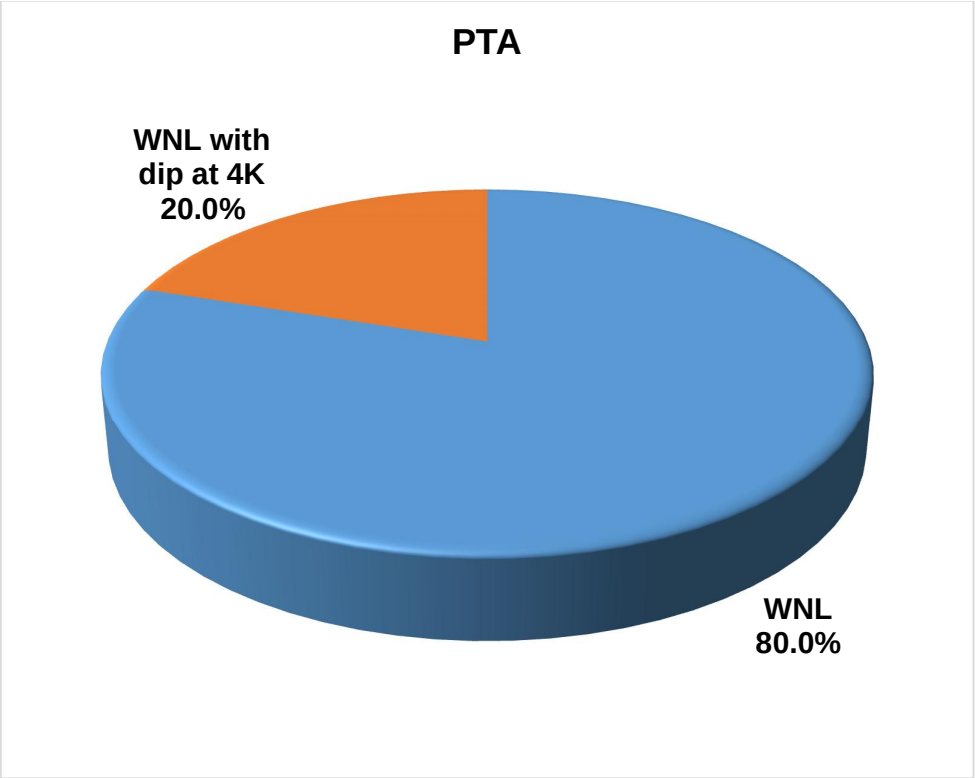
		N	%
Age group (years)	30 - 40 yrs	30	60.0
	> 40 yrs	20	40.0
	Total	50	100.0
PTA	WNL	40	80.0
	WNL with dip at 4K	10	20.0
	Total	50	100.0
IMPEDANCE	Normal	50	100.0
	Total	50	100.0
OAE	Absent	48	96.0
	Present	2	4.0
	Total	50	100.0
HIGH FREQUENCY	Absent	7	14.0
	Present	43	86.0
	Total	50	100.0

In my study, 60 % of my subjects belonged to age group of 30-40 year and 40% of cases are above 40 years. The pure tone audiogram of The cases are within normal limits. All patties had a normal impedance audiogram. The Otoacousitc emissions were absent in 96 % while it was present in Only 4 %.

Man Whitney test to compare between PTA groups

	PTA	N	Mean Rank	P-Value
RE-8K	WNL	40	25.15	0.043
	WNL with dip at 4K	10	34.90	
LE-8K	WNL	40	25.11	0.051
	WNL with dip at 4K	10	29.05	
RE-9K	WNL	40	24.68	0.04
	WNL with dip at 4K	10	34.80	
LE-9K	WNL	40	25.18	0.049
	WNL with dip at 4K	10	36.80	
RE-10.2K	WNL	40	23.30	0.028
	WNL with dip at 4K	10	34.30	
LE-10.2K	WNL	40	24.75	0.457
	WNL with dip at 4K	10	28.50	
RE-11.2K	WNL	40	21.80	<0.001
	WNL with dip at 4K	10	40.30	
LE-11.2K	WNL	40	22.85	0.009
	WNL with dip at 4K	10	36.10	
RE-12.5K	WNL	40	21.30	<0.001
	WNL with dip at 4K	10	42.30	
LE-12.5K	WNL	40	22.11	0.001
	WNL with dip at 4K	10	39.05	
RE-14K	WNL	40	22.24	0.001
	WNL with dip at 4K	10	38.55	
LE-14K	WNL	40	23.55	0.053
	WNL with dip at 4K	10	33.30	
RE-16K	WNL	40	24.23	0.224
	WNL with dip at 4K	10	30.60	
LE-16K	WNL	40	25.31	0.056
	WNL with dip at 4K	10	32.25	

This is the chart showing the P value relationship of normal PTA and PTA with dip at 4kHz each high frequency tones. In most case p value is < 0.05 and it is significant.



This picture shows cases with normal PTA and PTA with dip at 4kHz. 80% of cases shows normal PTA and 20.0% shows PTA with dip At 4 kHz

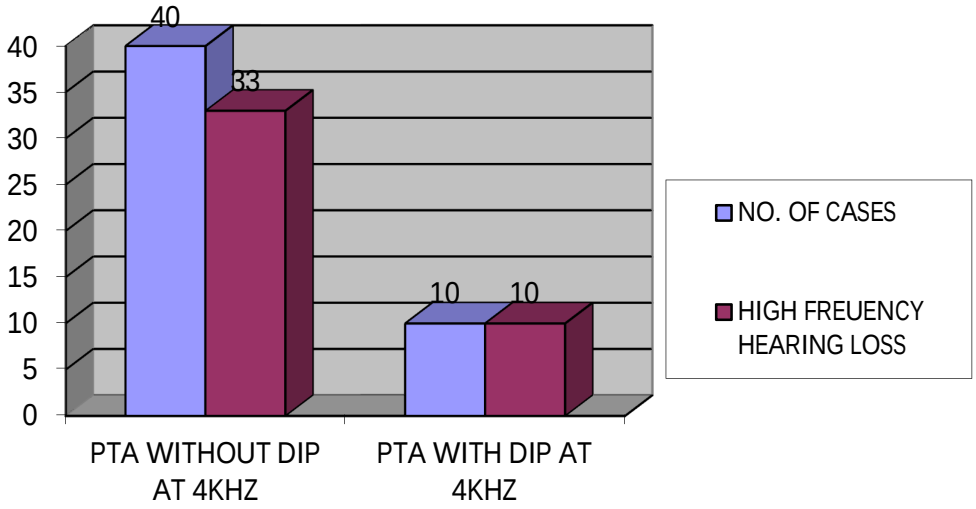
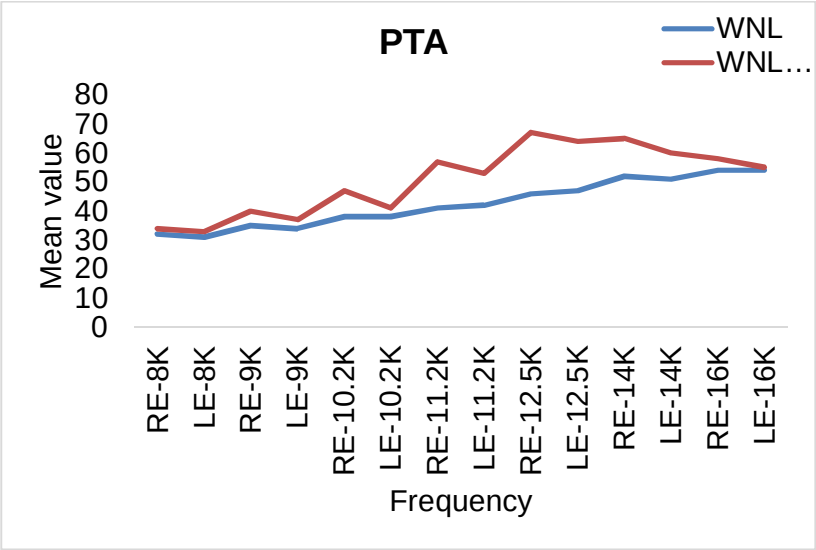


Figure 1 Relationship showing persons with normal PTA and PTA with dip at 4kHz to HF hearing loss

This Chart Shows that 82.5% (33 cases of 40) have a High Frequency Hearing Loss that is being misled by a normal PTA report whereas 100% of persons with dip at 4 KHz in PTA have a High Frequency Hearing Loss.



Comparison of Age Distribution and High Frequency Hearing loss Patterns

Age Group	No. Of Cases	Percentage with HF hearing loss
30- 40 yrs	30	83%
>40yrs	20	75%

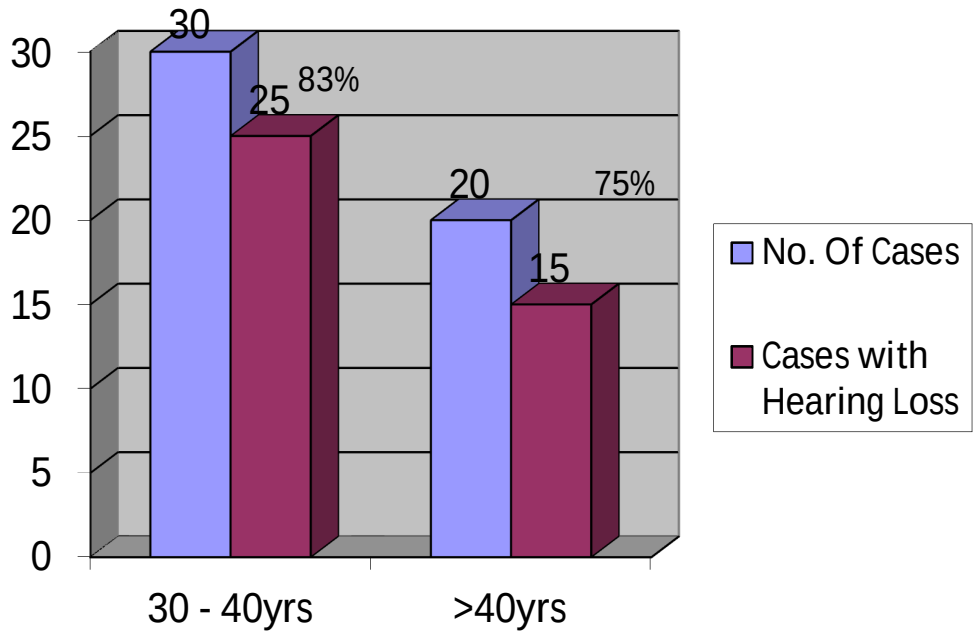
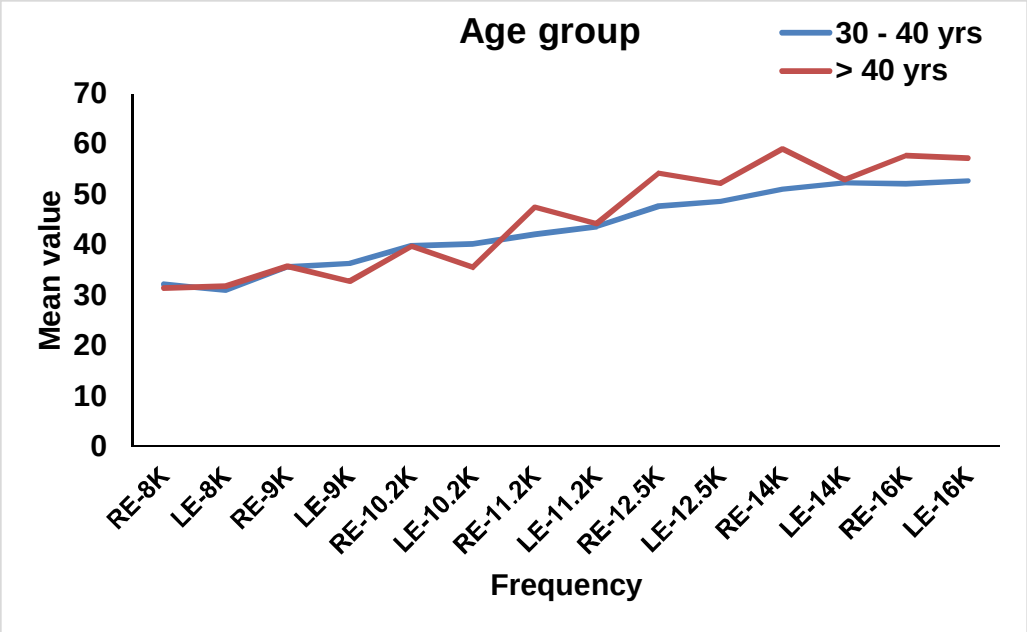


Figure 2 Comparison of Age distribution and High frequency Hearing Loss

83 % of subjects in the age group of 30-40 years have high frequency hearing loss while 75 % of subjects above 40 years of age have high frequency hearing loss.

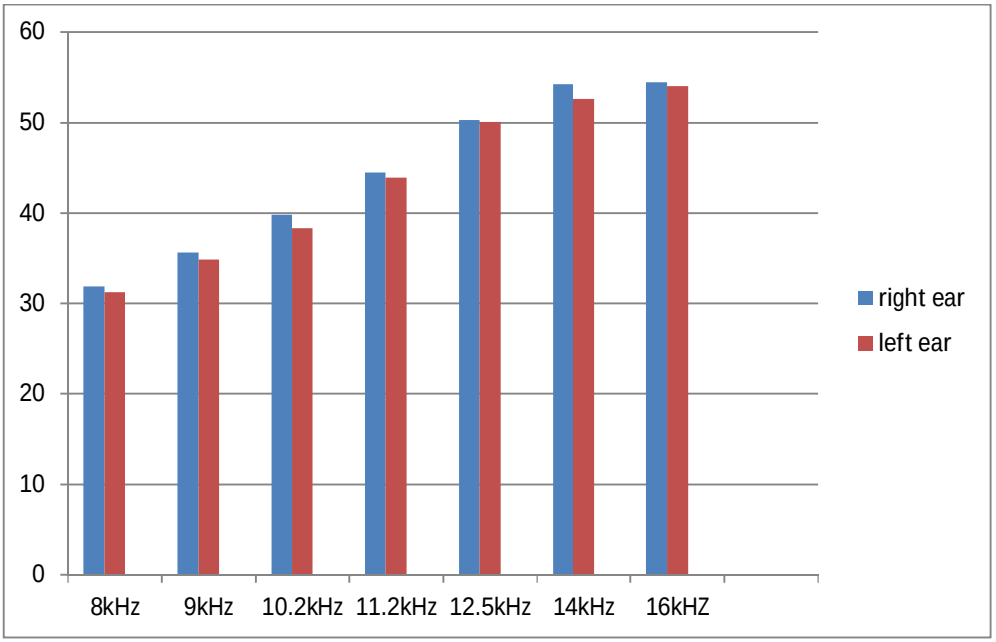
HEARING LOSS AT DIFFERENT FREQUENCIES AT DIFFERENT AGE GROUP



UNILATERAL / BILATERAL HEARING LOSS

Most of the subjects had symmetrical bilateral hearing loss in all high frequencies.

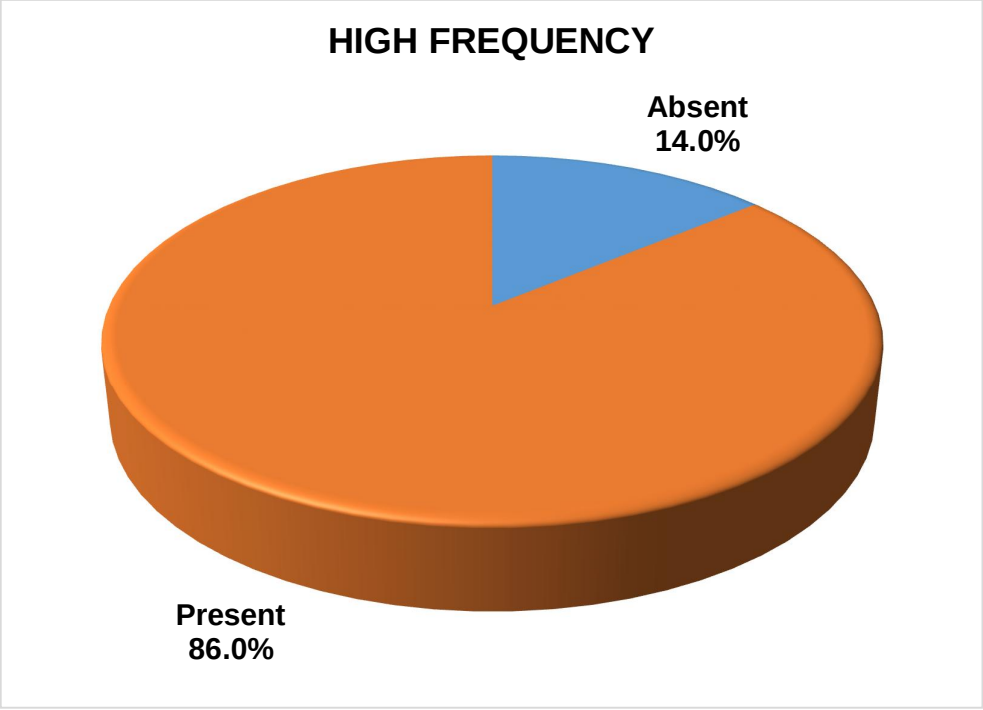
The bar diagram showing relationship high frequency hearing loss of right ear and left ear

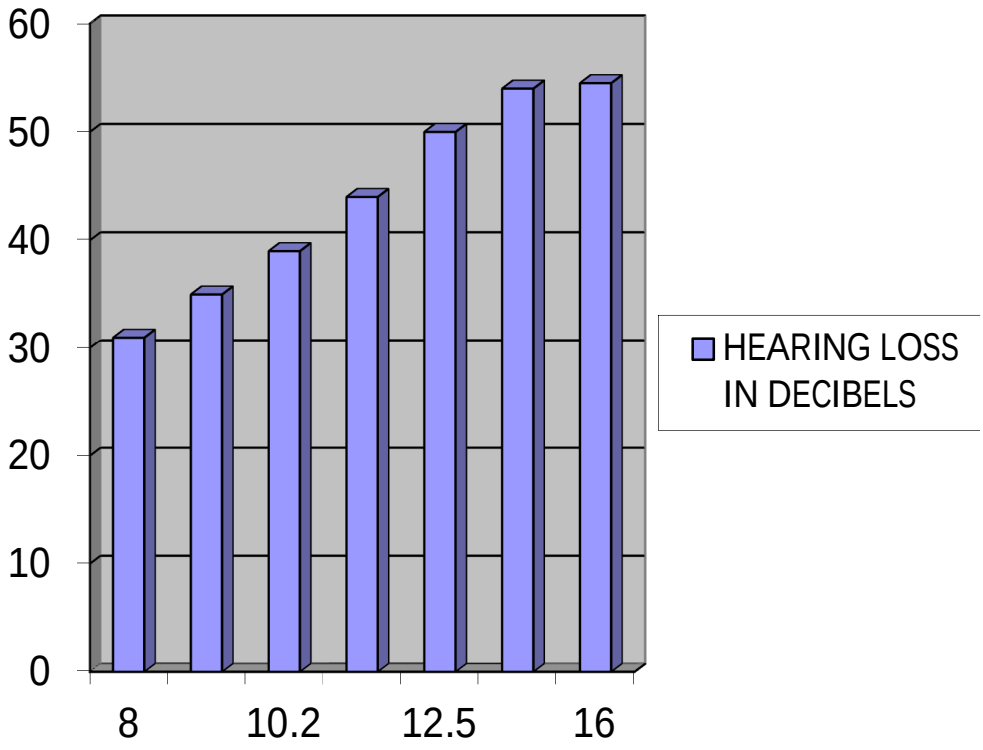


DESCRIPTIVE STATISTICS OF HIGH FREQUENCY PURE TONES HEARING LOSS FOR EACH FREQUENCY

Variables	Mean	Std. Dev	Median	1st Quartile	3rd Quartile
DURATION OF NOISE EXPOSURE	9.3	6.8	7.0	5.0	10.0
RE-8K	31.9	9.4	30.0	30.0	35.0
LE-8K	31.3	8.3	30.0	25.0	35.0
RE-9K	35.7	10.7	35.0	30.0	40.0
LE-9K	34.9	8.8	35.0	30.0	40.0
RE-10.2K	39.8	11.5	40.0	35.0	45.0
LE-10.2K	38.3	10.4	40.0	30.0	45.0
RE-11.2K	44.2	12.8	42.5	35.0	50.0
LE-11.2K	43.9	12.3	45.0	40.0	50.0
RE-12.5K	50.3	13.7	50.0	40.0	60.0
LE-12.5K	50.1	13.6	50.0	40.0	60.0
RE-14K	54.2	11.2	52.5	45.0	65.0
LE-14K	52.6	12.5	50.0	45.0	65.0
RE-16K	54.4	10.0	50.0	50.0	60.0
LE-16K	54.5	9.8	52.5	50.0	60.0

FIGURE SHOWING PERCENTAGE OF HIGH FREQUENCY HEARING LOSS

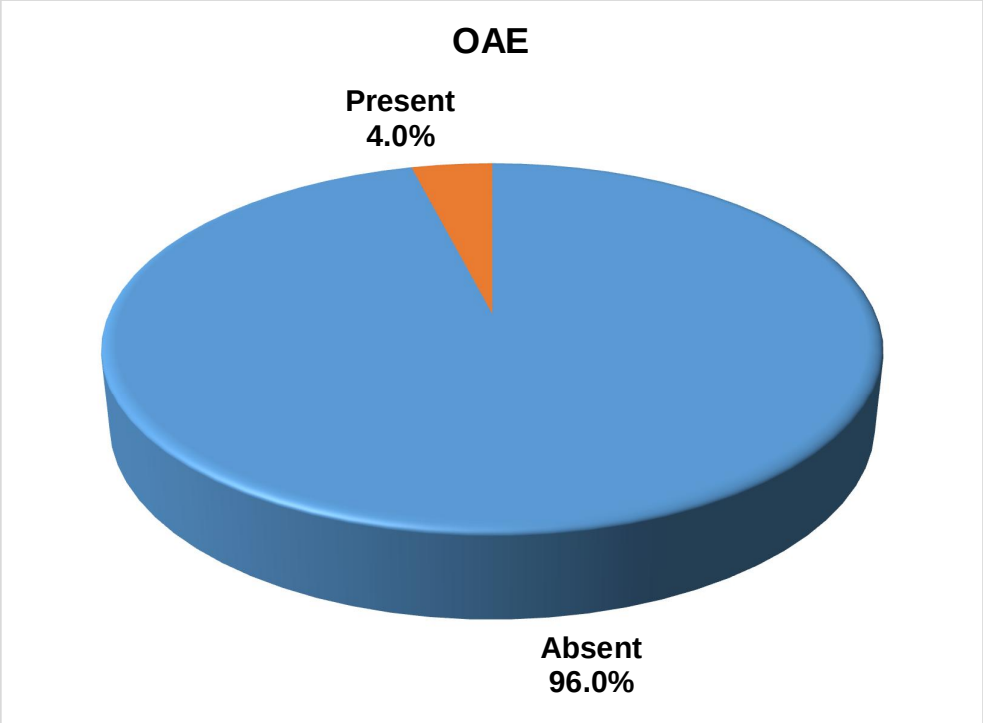




High frequency in kilo hertz

This is the bar diagram showing hearing loss in decibels at corresponding high frequencies.

This pie chart shows that 4 % of subjects have otoacoustic emissions. While 96 % have absent otoacoustic emissions. Those subjects with absent otoacoustic emissions have comparatively worse hearing loss high frequency than those with present otoacoustic emissions.



DURATION OF WORK

DUATION OF WORK	NO OF PATIENTS	PERCENTAGE
1-3	4	8
4-6	19	38
7-9	13	26
>10	14	28

This table shows duration of work in relation to the cases. 8 % of subjects worked in noisy surroundings for < 3 years, 38 % worked for 4 to 6 years, 26 % have worked for 7 – 9 years while 28 % have worked for > 10 years. Subjects who have worked for more than 4 Years have more significant hearing loss compared to those who have worked for less than 4 years.

CORRELATION BETWEEN DURATION OF WORK AND FREQUENCY

RE-8K	Correlation	-.098
	P-Value	.498
LE-8K	Correlation	-.081
	P-Value	.577
RE-9K	Correlation	.083
	P-Value	.568
LE-9K	Correlation	-.137
	P-Value	.344
RE-10.2K	Correlation	.335
	P-Value	.041
LE-10.2K	Correlation	.371
	P-Value	.025
RE-11.2K	Correlation	.323
	P-Value	.022
LE-11.2K	Correlation	.455
	P-Value	.052
RE-12.5K	Correlation	.389
	P-Value	.005
LE-12.5K	Correlation	.373
	P-Value	.008
RE-14K	Correlation	.460
	P-Value	.001
LE-14K	Correlation	.376
	P-Value	.042
RE-16K	Correlation	.377
	P-Value	.007
LE-16K	Correlation	.384
	P-Value	.006

This chart shows duration of work and each frequency affected. In most high frequencies the p value is < 0.05 and it is significant.

DISCUSSION

A prospective study to detect hearing loss in noise exposed individuals at an early stage prior to affecting speech frequency. The study was conducted in RGGGH, Chennai, MMC. Traffic Police and drivers of central zone are subjected to study. The average sound level of central zone is 109dB.No persons in the study have any hard of hearing as their compliant .All patient are subjected to conventional pure tone audiogram.

PTA.

Total No of Cases : 50

Normal- PTA : 40

PTA with dip at 4KHz: 10

Among the 40 patients with normal pure tone audiogram, 33 patients had high frequency hearing loss.82.5%cases had high frequency hearing loss with normal pure tone audiogram. The high frequency audiometry could detect hearing loss which was undetected by conventional pure tone audiometry. Thus we were able to diagnose hearing loss at an earlier stage before speech frequency was affected and

hence high frequency audiometry can be used as a preventive audiological tool.

100 % of patients with pure tone audiogram with dip at 4 kilo hertz developed high frequency hearing loss.

All patient had normal Tympanometry. All 50 have “A” type curve.

Ipsilateral and contralateral reflex present.

AGE DISTRIBUTION

83 % of subjects in the age group of 30-40 years have high frequency hearing loss while 75 % of subjects above 40 years of age have high frequency hearing loss.

DURATION OF EXPOSURE

Here we divide the work groups into four categories

- < 3 years
- 4 to 6 years
- 7 – 9 years
- 10 years

Subjects who have worked for more than 4 years have more significant hearing loss compared to those who have worked for less than 4 years.

UNILATERAL/ BILATERAL HEARING LOSS

Most of the subjects had symmetrical bilateral hearing loss in All high frequencies.

OTOACOUSTIC EMISSIONS

All the cases were subjected to Ot acoustic emissions and only 4 % cases showed presence of Otacoustic emissions while 96 % show absence of otoacoustic emissions. Since only 2 cases show presence of OAE statistical comparison is difficult .All subjects with high frequency hearing loss showed absence of otoacoustic emissions. A few patients with absent OAE had no high frequency hearing loss. We have to follow up these cases along with those showed presence of OAE .

HIGH FREQUENCY PURE TONE AUDIOGRAM

86% of the total patients had high frequency hearing loss who have normal pure tone audiogram.

INTERPRETATION OF RESULTS

A prospective study to detect hearing loss in those cases exposed to noisy environment before affecting speech frequency using high frequency audiometry. Study was conducted in RGGGH, MMC Chennai.

Traffic police and drivers of Chennai central were the subjects of study

- In my study 83% of subject in the age group 30-40years have high frequency hearing loss and 75% cases above the age 40 years have high frequency hearing loss

-82.5% of cases with normal PTA had high frequency hearing loss

-All the subjects with PTA with dip at 4kHz had high frequency hearing loss

-96.5 %of cases have absence of OAE

-There is considerable relation between duration of exposure and hearing loss and it is proven

-86% of the total cases have high frequency hearing loss with normal PTA.

-All most all cases have symmetrical bilateral hearing loss

BENEFITS TO THE COMMUNITY

- **T**his study helps in early detection of hearing loss those working in noisy environment before the speech frequency affected
- Helps in early intervention and prevention of NIHL
- Create awareness about noise exposure and hearing loss
- **I**mportance of wearing protective devices during work hours to prevent NIHL

DIFFERENT STUDIES AND THEIR INFERENCES

1.	D. A. Erickson et al	12-20 KHz
2.	S. A. Fausti et al	13-20 KHz
3.	Grzesik J et al (1983)	10-20 KHz
4.	Ahmed HO et al (2001)	10-18 KHz
5.	Turkkahraman S et al	4-16 KHz
6.	Serra et al (2005)	14 and 16 KHz

This table shows different studies conducted worldwide that demonstrates the predominantly affected frequency.

MY STUDY IS COMPARABLE WITH FOLLOWING STUDIES

"A study on early detection of noise induced hearing loss by using high frequency audiometry" by R.Singh, R.Saxena, S.Varshney Internet Journal of otorhinolaryngology showed detection of noise induced hearing loss in 62 % individuals using high frequency pure tone audiometry. 74% cases were detected in the younger age group.

"An article published on occup.med (London) 2001 June by Ahmedtio, Jerwoodo, Ballalsies et al. Showed that HFA can be used as an early indicator of noise induced hearing loss and acoustic trauma rather than conventional audiometry particularly for younger groups".

"A study on extended high-frequency audiometry in subjects exposed to occupational noise was conducted by stKorres GS, Ferekidis

E et al In Ear, Nose and Throat Department, National University of Athens, Hippokration Hospital, Greece published in B-ENT 2008. They conducted the study on 139 industry workers exposed to noise for a period of two years by using EHF audiometry in the frequency range 9-20 kHz. A statistically significant correlation was seqen between the elevation of puretone thresholds and time of exposure at all frequencies with the exception of 10,000 Hz."

"Study by soma G1 pictroiusti, magrini A. Coppetice et al in Department of occupational medicine toversata University, Italy, Published rencon journal of Industrial medicine 2008 study on 184 Cement workers showed EHFA is more sensitive than conventional audiometry in detecting noise based hearing loss, suggesting that EHFA could represent a useful preventive investigation."

"A study by Türkkahraman S, Gök U, Karlidağ T et al at Department of Otolaryngology, Kahramanmaraş State Hospital, Kahramanmaraş, Turkey and published in KBB = Journal of Ear, Nose, and Throat 2003 on hydroelectric power plant workers with no hearing problems suggested that high-frequency audiometry should be combined with standard audiometry in the detection and follow-up of individuals who are at potential risks for hearing losses".

"A study on application of extended high frequency audiometry in the early diagnosis of noise induced hearing loss by Wang Y, Yang B et al from Department of Otorhinolaryngology, Hospital of China First Automobile Group Corporation, Changchun 130011, China shows occurrence of threshold changes in the high frequency range is earlier than low frequency range. Ageing and working time also affect 10-20 Kilo Hertz hearing threshold."

A study on "Audiometric evaluation in extended high frequencies of individuals exposed to occupational noise by Porto MA, Gahyva DL et al from Departamento de Fonoaudiologia da Faculdade de Odontologia de Bauru da Universidade de São Pauloc demonstrated the contribution of high frequency audiogram for early diagnosis as these threshold are affected before conventional frequencies".

A study on "High frequency thresholds: variations with age and industrial noise exposure by Morton LP, Reynolds L from Department of Logopaedics, University of Cape Town published in The South African Journal of Communication Disorders showed significant differences in high frequency, thresholds between the noise exposed group and the normal subjects.

A study on “Extended high-frequency thresholds in noise-induced hearing loss by Hallmo P, Borchgrevink HM et al at Department of Otorhinolaryngology, Ullevål University Hospital, Oslo, Norway. Study on 167 male workers with history of noise exposure, shows age effect in the EHFA was present only in lowest grades of conventional frequency audiometry.”

A study on “Temporary threshold shift in military pilots measured using conventional and extended high-frequency audiometry after one flight by Kuronen P, Sorri MJ et al Finnish Air Force Headquarters, Finland International Journal of Audiology 2003 conducted study on 51 Finnish Air Force military personnel as subjects using HFA and conventional pure tone audiometry. A statistically significant temporary threshold shifts (TTS) at several frequencies and with all aircraft types involved was noted”.

A study on “High-frequency audiometry: a means for early diagnosis of noise-induced hearing loss by Mehrparvar AH, Mirmohammadi SJ at Department of Occupational Medicine, Shahid Sadoughi University of Medical Sciences, Yazd, Iran, pulished in Noise & Health 2011 compared hearing threshold and prevalence of hearing loss in conventional and high frequencies of audiometry among

textile workers with and without exposure to noise more than 85 dB. The hearing threshold was significantly higher at 16000 Hz ”

A study on “High-frequency audiometry- Normative studies and preliminary experiences by Laukli E, Mair IW published in Scandinavian Audiology 1985 on high-frequency audiometer and its use for measuring hearing thresholds between 8 and 20 kHz. It showed that audiometer can be used in the clinic with approximately the same degree of reproducibility as in conventional audiometry.”

A study on “High-frequency (10-18 kHz) hearing thresholds: reliability, and effects of age and occupational noise exposure by Dennis J H , Badran O, Ismail M et al from Department of Family and Community Medicine, College of Medicine, King Faisal University, Dammam, Saudi Arabia published a journal 2001 showed that high-frequency audiometry (HFA) was as reliable as the conventional procedure.”

A study on “Extended high frequency audiometry in the prevention of noise induced hearing loss by Somma G, Coppeta L, Magrini A et al from Giornale Italiano di Medicina del Lavoro ed Ergonomia 2007.

They conducted a cross-sectional study involving 204 industrial noise exposed and 100 non-industrial noise-exposed workers which showed significant differences at EHFA were detected noise-exposed workers with normal findings at conventional audiometry

A study by turkkahraman S, Gok U, Ozturk A etal., Department of otorhinolaryngology Kahranabnaras State Hospital Turkey Journal of Ear, Nose, Throat 2003 10(4) 137-142 study on 64 male workers suggested that high-frequency audiometry should be combined with Standard audiometry in the detection and follow-up of individuals who are at potential risks for hearing losses".

CONCLUSION

From my study the following conclusions were made. unprotected Exposure to loud noise for a long period of time produce hearing Impairment. Initially it affects high frequencies and gradually progress to low frequency.

By the time hearing loss is detected using conventional audiometry damage has already been affect the speech frequencies. It will affect the verbal communication of the patient and affect the quality of Life .Therefore by using high frequency audiometry early detection of hearing loss in the high frequency can be detected. It will help us to take an early warning to those working in noisy environment to take preventive measures.

The high frequency pure tone audiometry helps to diagnosis early noise induced hearing loss and for the assessment of the susceptibility of the individuals to noise damage. The high frequency audiometry can be used as a preventive and interventional method to prevent NIHL. Hereby to improve the quality of social life.

There is considerable relation between duration of exposure and hearing loss and it is proven.

Majority of patients had bilateral symmetrical hearing loss. No asymmetrical hearing loss noted.

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ABBREVIATION

NIHL	NOISE INDUCED HEARING LOSS
TTS	TEMPORARY THRESHOLD SHIFT
PTS	PERMANENT THRESHOLD SHIFT
CN	COCHLEAR NUCLEI
AVCN	ANTEROVENTRAL COCHLEAR NUCLEI
PVCN	POSTERO VENTRAL COCHLEAR NUCLEI
SOC	SUPERIOR OLIVARY COMPLEX
PTA	PURE TONE AUDIOMETRY
HFA	HIGH FREQUENCY AUDIOMETRY
SPL	SOUND PRESSURE LEVEL
IHC	INNER HAIR CELL
OHC	OUTER HAIR CELL
OAE	OTOACOUSTIC EMISSION
HOH	HARD OF HEARING
WHO	WORLD HEALTH ORGANISATION
ROS	REACTIVE OXYGEN SPECIES
PHC	PRIMARY HEALTH CENTER

PROFORMA

HISTORY OF THE PATIENT

NAME :

AGE:

SEX:

OCCUPATION, PERIOD OF JOB:

HOURS OF WORK PER DAY:

ANY EAR COMPLAINTS: YES NO

IF YES

HARD OF HEARING EAR DISCHARGE :

RINGING SENSATION OF EAR:

GIDDINESS:

ANY CO MORBIDITIES: YES NO

IF YES

1.FAMILY HISTORY OF HEARING LOSS

SMOKING

HYPERTENSION

DIABETES MELITUS

THYRIOD DISORDERS

HISTORY OF ANY DRUG INTAKE

ENT EXAMINATION

EAR :

NOSE

THROAT:

TUNIC FORK TEST:

Rinne :

Weber:

ABC :

PURE TONE AUDIOMETRY :

IMPEDANCE AUDIOGRAM :

ACOUSTIC REFLEX :

TYMPANOGRAM

OTOACOUSTIC EMISSION:

HIGH FREQUENCY AUDIOGRAM

CONSENT FORM

STUDY TITLE : A STUDY ON "ASSESSMENT OF HEARING LOSS IN HIGH RISK INDIVIDUALS USING HIGH FREQUENCY PURE TONE AUDIOMETRY"

I …… hereby give consent to participate in the study conducted by Dr.MANJU JOSEPH, Post Graduate in Upgraded Institute of Otorhinolaryngology, Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations.

Signature / Thumb impression of the patient / relative Place Date

Patient Name and Address

Signature of the Investigator

Signature of the Guide

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.Manju Joseph,
PG in MS ENT,
Upgraded Institute of Otorhinolaryngology,
MMC & RGGGH, Chennai -3

Dear Dr.Manju Joseph,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Assessment of hearing loss in high risk Individuals using high frequency pure tone audiometry" No.05042013.

The following members of Ethics Committee were present in the meeting held on 17.04.2013 conducted at Madras Medical College, Chennai -3.

1. Dr.G.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. Kalai Selvi MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3 (Incharge)
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. A. Radhakrishnan MD -- Member
   Prof of Internal Medicine, MMC, Ch-3
6. Prof. S. Deivanayagam MS -- Member
   Prof of Surgery, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

18/4/13

Member Secretary, Ethics Committee

MASTER CHART

SL. NO.	AGE	SEX	DURATION	PTA	IMPEDANCE	OAE	High frequency hearing loss in dB	8kHz RE,LE	9kHz RE,LE	10.2kHz RE ,LE	11.2kHz RE,LE	12.5kHz RE,LE	14kHz RE,LE	16kHz RE,LE
			OF NOISE											
	Sl. No.		EXPOSURE											
1	48	M	7 Yrs	WNL	NORMAL STUDY	ABSENT	present	30,40	40,50	30,40	40,50	50,60	50,60	60,60
2	37	M	7	WNL	NORMAL STUDY	ABSENT	present	35,25	35,30	20,20	25,30	40,40	65,65	70,70
3	31	M	6	WNL	NORMAL STUDY	ABSENT	present	30,30	30,35	40,40	35,35	45,40	40,45	45,40
4	40	M	6	WNL	NORMAL STUDY	ABSENT	present	50,,40	40,40	40,50	40,50	50,50	40,45	40,50
5	36	M	6	WNL	NORMAL STUDY	ABSENT	present	30,35	40,40	35,30	40,40	45,45	50,50	55,55
6	39	M	8	WNL	NORMAL STUDY	ABSENT	present	30,35	40,45	45,50	40,55	50,55	55,50	40,50
7	38	M	7	WNL	NORMAL STUDY	ABSENT	present	35,35	40,45	45,45	45,50	45,55	55,50	55,60
8	38	M	7	WNL	NORMAL STUDY	ABSENT	presnt	30,30	30,45	40,45	40,40	50,60	50,50	50,55
9	40	M	8	WNL	NORMAL STUDY	ABSENT	absent	10,25	15,30	15,20	25,20	20,20	15.25	30,25

10	47	M	6	WNL	NORMAL STUDY	ABSENT	present	30,30	40,45	50,60	65,75	65,75	70,70	50,50
11	42	M	10	WNL	NORMAL STUDY	ABSENT	present	45,40	45,30	40,30	50,50	60,60	65,65	70,70
12	47	M	6	WNL	NORMAL STUDY	ABSENT	present	30,30	25,20	30,35	40,40	40,50	50,45,	60,60
13	46	M	8	WNL	NORMAL STUDY	ABSENT	present	30,40	40,30	40,35	30,40,	35,40	45,50	50,50
14	35	M	15	WNL	NORMAL STUDY	ABSENT	absent	25,10	25,25	25,25	25,30	20,25	25,25	25,25
15	39	M	5	WNL	NORMAL STUDY	ABSENT	pesent	30,25	25,35	35,40	45,45	45,45	60,70	50,50
16	46	M	10	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	35,40	35,30	45,45	65,60	70,75	70,70	70,70
17	49	M	10	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	60,30	70,40	65,40	65,40	75,40	70,35	50,40
18	44	M	5	WNL	NORMAL STUDY	ABSENT	absent	35,25	20,25	25,25	35,35	20,15	15,15	25,25
19	50	M	5	WNL	NORMAL STUDY	ABSENT	absent	30,30	35,35	20,25	25,25	15,15	15,25	15.2
20	38	M	4	WNL	NORMAL STUDY	ABSENT	present	15,15	35,20	30,15	35,20	35,20	4,35	50,45
21	54	M	33	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	30,35	40,35	55,45	60,70	65,75	65,70,,	,70,70
22	43	M	7	WNL	NORMAL STUDY	ABSENT	present	30,35	35,40	35,45	40,45	45,40	45,50	55,50
23	38	M	18	WNL	NORMAL STUDY	ABSENT	present	25,30	35,35	40,35	45,45	50,55	55,50	45,55

24	35	M	10	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	30,30	30,35	40,50	50,65	70,80	70,70	60,60
25	33	M	3	WNL	NORMAL STUDY	ABSENT	present	30,35	40,45	45,40	45,45	45,50	55,50	55,60
26	31	M	10	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	30,25	35,35	50,40	55,55	70,65	70,70	70,55
27	45	M	21	WNL	NORMAL STUDY	ABSENT	present	25,15	30,20	60,20	60,35	70,50	70,50	70,70
28	50	M	15	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	35.3	50,45	65,40	80,60	70,60	60,50	60,50
29	37	M	18	WNL	NORMAL STUDY	ABSENT	present	35,35	40,40	45,40	45,45	50,50	50,60	70,70
30	39	M	10	WNL	NORMAL STUDY	ABSENT	present	25,30	25,20	40,40	55,50	60,55	50,50	70,70
31	65	M	30	WNL WITH DIP AT 4K	NORMAL STUDTY	ABSENT	present	25,30	35,30	40,30	55,35	80,80	70,70	50,50
32	38	M	7	WNL	NORMAL STUDY	ABSENT	present	65,20	75,30	70,30	80,40	80,50	70,70	50,50
33	37	M	20	WNL	NORMAL STUDY	ABSENT	present	30 ,25	35,25	30,30	35,30	35,55	70,75	70,70
34	46	M	6	WNL	NORMAL STUDY	ABSENT	absent	20,15	25,15	25,15	30,10	25,25	20,20	NR,NR
35	41	M	15	WNL	NORMAL STUDY	ABSENT	absent	25,15	25,20	20,15	30,20	15,20	5015	25,25
36	49	M	25	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	30,45	40,45	40,45	50,50	60,55	55,50	50,50
37	27	M	3	WNL	NORMAL STUDY	ABSENT	present	30,35	35,40	40,45,	35,45	40,45	45,45	50,50

38	43	M	5	WNL	NORMAL STUDY	ABSENT	present	35,35	40,45	45,45	50,55	50,60	65,60	55,60
39	25	M	10	WNL	NORMAL STUDY	ABSENT	present	40,40	40,45	50,50	45,40	50,55	50,55	60,55
40	48	M	5	WNL	NORMAL STUDTY	absent	present	30,40	15,20	20,25	25,25	60,30	60,45	50,55
41	26	M	4	WNL	NORMAL STUDTY	ABSENT	present	35,40	45,40	40,45	50,55	50,45	45,40	45,40
42	28	M	4	WNL WITH DIP AT 4K	NORMAL STUDY	ABSENT	present	40,30	35,35	45,40	46,50	55,50	45,45	50,55
43	33	m	1	WNL	NORMAL STUDY	present	absent	20,25	20,40	15,60	20,60	25,15	30,25	25,20
44	43	M	7	WNL WITHDIP AT 4K	NORMAL STUDY	ABSENT	present	20,35	30,35	25,35	40,45	50,60	70,70	50,50
45	35	m	6	WNL	NORMAL STUDY	absent	present	35,45	40,45	40,45	45,40	40,45	45,45	4545
46	30	m	5	WNL	NORMAL STUDY	ABSENT	present	40,40	35,30	45,45	40,45	50,40	45,45	40,45
47	38	m	4	WNL	NORMAL STUDY	absent	present	30,30	35,35	40,40	45,45	40,40	40,40	40,35
48	35	m	6	WNL	NORMAL STUDY	ABSENT	present	35,35	40,45	40,45	35,40	45,50	50,50	45,45
49	30	M	6	WNL	NORMAL STUDY	ABSENT	present	40,45	40,40	40,45	45,40	50,50	55,50	60,55
50	34	m	4	WNL	NORMAL STUDY	PRESENT	present	30,30	35,40	45,40	30.25	40,35	35,35	40,35

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E-mail	dr.manjujoseph@gmail.com
Submission time	22-Dec-2013 09:58PM
Total words	10018

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INTRODUCTION Exposure to loud noise is the most common cause of sensori neural hearing loss in adults. It is an irreversible type of hearing loss typically affecting higher frequency initially then gradually progressing to lower frequency. NIHL affects day to day life, leads to social isolation and impaired communication. NIHL is a preventable type of hearing loss through early interventions. The magnitude of hearing loss is related to duration, intensity and nature of exposure 1 .NIHL involves all the cellular systems of cochlea. Hearing impairment is the loss of ability to detect certain frequencies of sound. NIHL can be permanent or temporary .Single exposure to loud noise leads to an...